

EXHIBIT 2

Confidential Subject to Protective Order

1 UNITED STATES DISTRICT COURT
2 SOUTHERN DISTRICT OF NEW YORK

3 IN RE: ACETAMINOPHEN -) MDL No. 3043
4 ASD-ADHD PRODUCTS)
5 LIABILITY LITIGATION) Case No.
6) 1:22-md-03043-DLC
7 THIS DOCUMENT RELATES TO:)
8) JUDGE DENISE
9 All Cases, 1:22-md-03043) COTE

10 WEDNESDAY, AUGUST 30, 2023

11 CONFIDENTIAL - PURSUANT TO PROTECTIVE ORDER

12 - - -

13 Videotaped deposition of Wendy
14 Chung, MD, Ph.D., held at the offices of
15 Barnes & Thornburg, One Marina Park Drive,
16 Suite 1530, Boston, Massachusetts, commencing
17 at 8:42 a.m. Eastern, on the above date,
18 before Carrie A. Campbell, Registered
19 Diplomate Reporter, Certified Realtime
20 Reporter, Illinois, California & Texas
21 Certified Shorthand Reporter, Missouri,
22 Kansas, Louisiana & New Jersey Certified
23 Court Reporter.

24 - - -

25 GOLKOW LITIGATION SERVICES
877.370.DEPS
deps@golkow.com

1 A P P E A R A N C E S :

2
3 TRACEY & FOX
4 BY: SEAN P. TRACEY
5 stracey@traceylawfirm.com
6 LAWRENCE TRACEY
7 ltracey@traceylawfirm.com
8 440 Louisiana Street, Suite 1901
9 Houston, Texas 77002
10 (713) 495-2333

11 and

12 THE LANIER LAW FIRM, PLLC
13 BY: EVAN M. JANUSH (VIA ZOOM)
14 evan.janush@lanierlawfirm.com
15 CATHERINE HEACOX (VIA ZOOM)
16 catherine.heacox@lanierlawfirm.com
17 126 East 56th Street, 6th Floor
18 New York, New York 11758
19 (212) 421-2800

20 and

21 KELLER POSTMAN LLC
22 BY: REBECCA KING (VIA ZOOM)
23 rebecca.king@kellerpostman.com
24 ASHLEY C. KELLER (VIA ZOOM)
25 ashley.keller@kellerpostman.com
26 ASHLEY BARRIERE (VIA ZOOM)
27 ashley.barriere@kellerpostman.com
28 AMANDA HUNT (VIA ZOOM)
29 amanda.hunt@kellerpostman.com
30 ROSIE ROMANO (VIA ZOOM)
31 rosie.romano@kellerpostman.com
32 LAUREN SCHULTZ (VIA ZOOM)
33 lauren.schultz@kellerpostman.com
34 J.J. SNIDOW (VIA ZOOM)
35 jj.snidow@kellerpostman.com
36 150 North Riverside Plaza, Suite 4100
37 Chicago, Illinois 60606
38 (312) 741-5220

1 and

2

WATTS GUERRA LLC

3 BY: MIKAL C. WATTS

mcwatts@wattsguerra.com

4 HAILEY WATTS

hwatts@wattsguerra.com

5 RUSS ABNEY (VIA ZOOM)

rabney@wattsguerra.com

6 JOHN CRACKEN (VIA ZOOM)

jcracken@wattsguerra.com

7 Millennium Park Plaza RFO

Suite 410, C112

8 Guaynabo, Puerto Rico 00966

(210) 447-0500

9

10 and

11

HOLWELL SHUSTER & GOLDBERG LLP

12 BY: EILEEN MONAGHAN DELUCIA (VIA ZOOM)

edelucia@hsgllp.com

13 425 Lexington Avenue

New York, New York 10017

14 (646) 837-5151

15

and

16

17 MOSKOW LAW GROUP LLC

BY: NEAL L. MOSKOW

18 neal@moskowlaw.com

425 Kings Highway East

19 Fairfield, Connecticut 06825

(475) 999-4177

20

21 and

22

WAGSTAFF & CARTMELL

23 BY: LINDSEY SCARCELLO (VIA ZOOM)

lscarcello@wcllp.com

24 4740 Grand Avenue, Suite 300

Kansas City, Missouri 64112

25 (816) 701-1100

1 and

2

THE CARLSON LAW FIRM

3 BY: EMILY MARLOWE (VIA ZOOM)

emarlowe@carlsonattorneys.com

4 1717 North Interstate Highway 35,
Suite 305

5 Round Rock, Texas 78664
6 (512) 671-7277

7 and

8

KRAUSE & KINSMAN

9 BY: TRICIA CAMPBELL (VIA ZOOM)

tcampbell@krauseandkinsman.com

10 4717 Grand Avenue, Suite 300

Kansas City, Missouri 64112

11 (816) 200-2900

12

and

13

14 DOVEL & LUNER

BY: JULIEN ADAMS (VIA ZOOM)

15 julien@dovel.com

GREG DOVEL (VIA ZOOM)

16 greg@dovel.com

201 Santa Monica Boulevard, Suite 600

17 Santa Monica, California 90401

(310) 656-7066

18

19 and

20

TORHOERMAN LAW LLC

21 BY: ERIC CRACKEN (VIA ZOOM)

ecracken@thlawyer.com

22 227 West Monroe Street, Suite 2650

Chicago, Illinois 60606

23 (312) 372-4800

24

and

25

1 BEASLEY, ALLEN, CROW, METHVIN,
PORTIS & MILES
2 BY: JASON SHORT (VIA ZOOM)
jason.short@beasleyallen.com
3 218 Commerce Street
Montgomery, Alabama 36104
4 (800) 898-2034

5
6 and

7 KERSHAW TALLEY BARLOW
BY: WILLIAM J. LEE (VIA ZOOM)
8 VINH T. LE (VIA ZOOM)
401 Watt Avenue, Suite 1
9 Sacramento, California 95864-7273
(916) 520-6639

10
11 and

12 COOPER LAW PARTNERS
BY: DAVIS COOPER (VIA ZOOM)
13 davis@cooperlawpartners.com
999 Vanderbilt Beach Road, Suite 200
14 Naples, Florida 34108
(800) 872-3500

15
16 and

17 COOPER & KIRK PLLC
18 BY: JOSEPH O. MASTERMAN (VIA ZOOM)
jmasterman@cooperkirk.com
19 523 New Hampshire Avenue NW
Washington, DC 20036
20 (202) 220-9600
Counsel for Plaintiffs

21
22
23
24
25

1 SKADDEN, ARPS, SLATE, MEAGHER & FLOM LLP
2 BY: ALLISON M. BROWN
allison.brown@skadden.com
3 One Manhattan West
New York, New York 10001
(212) 735-3000
4

5 and

6 BARNES & THORNBURG LLP
7 BY: JAMES F. MURDICA (VIA ZOOM)
jmurdica@btlaw.com
8 2029 Century Park East, Suite 300
Los Angeles, California 90067-2904
9 (310) 284-3880

10 and
11

12 BARNES & THORNBURG LLP
13 BY: DEANNA LEE (VIA ZOOM)
dlee@btlaw.com
14 555 12th Street N.W., Suite 1200
Washington, DC 20004-1275
15 (202) 289-1313

16 and

17 BARNES & THORNBURG LLP
18 BY: JESSICA BRENNAN (VIA ZOOM)
jessica.brennan@btlaw.com
19 67 East Park Place, Suite 500
Morristown, New Jersey 07960
20 (973) 775-6101

21 and
22

23

24

25

1 BARNES & THORNBURG, LLP
2 BY: KARA KAPKE (VIA ZOOM)
3 kara.kapke@btlaw.com
4 11 South Meridian Street
5 Indianapolis, Indiana 46204
6 (317) 236-1313

7 and

8 BUTLER SNOW
9 BY: DAVID M. COHEN (VIA ZOOM)
10 david.cohen@butlersnow.com
11 RAQUEL LUCAS (VIA ZOOM)
12 raquel.lucas@butlersnow.com
13 810 7th Avenue, Suite 1105
14 New York, New York 10019
15 (646) 606-2996
16 Counsel for Johnson & Johnson
17 Consumer, Inc.

18 BARNES & THORNBURG LLP
19 BY: SANDRA M. KO (VIA ZOOM)
20 sko@btlaw.com
21 555 12th Street N.W., Suite 1200
22 Washington, DC 20004-1275
23 (202) 289-1313
24 Counsel for Costco Wholesale
25 Corporation

ARNOLD & PORTER, LLP
BY: RAYNE ELLIS (VIA ZOOM)
rayne.ellis@arnoldporter.com
250 West 55th Street
New York, New York 10019
(212) 836-8000
Counsel for Dollar Tree Inc.,
7-Eleven, and Family Dollar, Inc.

1 KING & SPALDING LLP
2 BY: EVA CANAAN (VIA ZOOM)
3 ecanaan@kslaw.com
4 1185 Avenue of the Americas
5 New York, New York 10036
6 (212) 556-2100

7 and

8 KING & SPALDING LLP
9 BY: LUKE BOSSO (VIA ZOOM)
10 lbosso@kslaw.com
11 1700 Pennsylvania Avenue NW
12 Washington, DC 20006
13 (202) 737-0500
14 Counsel for Walmart Inc., and
15 Wal-Mart Stores, Inc.

16

17 MORRISON & FOERSTER LLP
18 BY: LYNDESEY CAIN (VIA ZOOM)
19 lcain@mofo.com
20 ASHLEY E. QUINN (VIA ZOOM)
21 aquinn@mofo.com
22 250 West 55th Street
23 New York, New York 10019-9601
24 (212) 468-8000
25 Counsel for Target Corporation

17 DUANE MORRIS LLP
18 BY: SEAN K. BURKE (VIA ZOOM)
19 sburke@duanemorris.com
20 901 New York Avenue N.W., Suite 700 East
21 Washington, DC 20001-4795
22 (202) 776-5236
23 Counsel for Dollar General, Dollar
24 General Corporation
25

1 SMITH SOVIK KENDRICK & SUGNET
2 BY: DAVID M. KATZ (VIA ZOOM)
3 dkatz@smithsovik.com
4 250 South Clinton Street, Suite 600
5 Syracuse, New York 13202
6 (315) 474-2911
7 Counsel for Rite Aid

8
9 STONE DEAN LLP
10 BY: JOSEPH A. LARA (VIA ZOOM)
11 jlara@stonedeanlaw.com
12 21052 Oxnard Street
13 Woodland Hills, California 91367
14 (818) 999-2232
15 Counsel for The Kroger Co.

16
17 HAIGHT BROWN & BONESTEEL LLP
18 BY: KEITH ROZANSKI (VIA ZOOM)
19 krozanski@hbblaw.com
20 555 South Flower Street, 55th Floor
21 Los Angeles, California 90071
22 (213) 542-8000
23 Counsel for Big Lots Stores-PNS, LLC

24
25 ALSO PRESENT:
26 DANIEL OLIVO, Tracey Fox & Walters
27
28 MICHAEL KAUFFMANN, trial technician,
29 Precision Trial Solutions
30
31 LISA QIAN, firm unknown
32
33 NAO TAKADA, firm unknown

34
35 V I D E O G R A P H E R :
36 ROBERT MARTIGNETTI,
37 Golkow Litigation Services

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1 VIDEOGRAPHER: We are now on
2 the record. My name is Robert
3 Martignetti. I'm a videographer for
4 Golkow Litigation Services.

5 Today's date is August 30,
6 2023, and the time is 8:42 a.m.

7 This video deposition is being
8 held in Boston, Massachusetts, In Re:
9 Acetaminophen/ASD-ADHD Products
10 Liability Litigation.

11 The deponent is Wendy Chung,
12 MD, Ph.D.

13 Counsels' appearances will be
14 noted on the stenographic record.

15 The court reporter is Carrie
16 Campbell and will now swear in the
17 witness.

18
19 WENDY CHUNG, MD, Ph.D.,
20 of lawful age, having been first duly sworn
21 to tell the truth, the whole truth and
22 nothing but the truth, deposes and says on
23 behalf of the Plaintiffs, as follows:

24 /

25 /

1 DIRECT EXAMINATION

2 QUESTIONS BY MR. TRACEY:

3 Q. Dr. Chung, could you please
4 introduce yourself?

5 A. Sure. I'm Wendy Chung.

6 Q. And where do you live?

7 A. 20 Village Way, Brookline,
8 Massachusetts.

9 Q. And, Dr. Chung, my name is Sean
10 Tracey. You and I met before the deposition,
11 right?

12 A. That's correct.

13 Q. And do you understand I'm a
14 lawyer who's going to ask you questions in
15 this case?

16 A. Yes.

17 Q. One of the questions I want to
18 ask you is how you got hired in this case.

19 Can you tell me that?

20 A. I was approached by Eva Canaan.

21 Q. Who?

22 A. Eva Canaan.

23 Q. Who is Eva Canaan?

24 A. She's a lawyer.

25 Q. With what firm?

1 A. I don't recall. You -- I'm
2 sure it's easy to look up.

3 **Q. How do you know her?**

4 A. I had worked with her
5 previously.

6 **Q. On what kind of case?**

7 A. Another legal case in terms of
8 trying to understand a product and whether or
9 not a product was responsible for an outcome.

10 **Q. What product was that?**

11 A. Zofran.

12 **Q. Zofran?**

13 A. Yes.

14 **Q. Was that a Zofran birth defect**
15 **case?**

16 A. It was.

17 **Q. And is that a GSK product,**
18 **Zofran? I've forgotten.**

19 A. I don't recall who manufactures
20 Zofran.

21 **Q. Okay. Did you give a**
22 **deposition in that case?**

23 A. Yes.

24 **Q. All right. Did you testify at**
25 **trial?**

1 A. I did not.

2 Q. I've read some of your
3 depositions. I got ahold of three or four of
4 them, and I know you've testified in other
5 pharmaceutical litigation?

6 A. Yes, I have.

7 Q. You testified in, you told us,
8 the Zofran litigation. I think you
9 testified --

10 A. Let me --

11 Q. Pardon me?

12 A. I just want to clarify. I gave
13 deposition. I haven't testified.

14 Q. Yeah. Good point.

15 So we lawyers call deposition
16 testimony.

17 A. Ah, okay.

18 Q. It's the same kind of testimony
19 if you were testifying live at trial.

20 Do you understand that?

21 A. Okay.

22 Q. So they make you raise your
23 right hand. You swear to tell the truth.

24 This testimony is just as
25 binding as if you were to stare at a jury in

1 **the eye.**

2 **Okay?**

3 A. I understand.

4 MS. BROWN: I object to that.

5 QUESTIONS BY MR. TRACEY:

6 **Q. So when we say "testimony,"**
7 **lawyers, we mean both.**

8 **Okay?**

9 A. Understood.

10 **Q. So you have given sworn**
11 **testimony in other drug cases as I understand**
12 **it?**

13 MS. BROWN: Objection to the
14 form.

15 You can answer.

16 THE WITNESS: I had give a
17 dep -- given a deposition in the
18 Zofran litigation.

19 QUESTIONS BY MR. TRACEY:

20 **Q. And didn't you give a**
21 **deposition in an SSRI case?**

22 A. I did.

23 **Q. What other drug or device cases**
24 **have you testified in?**

25 A. Those are the only two.

1 **Q. The only two.**

2 **Have you consulted before this**
3 **case for Johnson & Johnson or any of the**
4 **affiliated Johnson & Johnson companies?**

5 A. No, I have not.

6 **Q. Okay. Do you consult for drug**
7 **companies outside of litigation?**

8 A. I am --

9 MS. BROWN: Object to the form
10 of the question.

11 Go ahead.

12 THE WITNESS: I am on
13 scientific advisory boards or boards
14 of directors.

15 QUESTIONS BY MR. TRACEY:

16 **Q. How many companies are you on**
17 **the scientific advisory boards or boards of**
18 **directors?**

19 A. I would have to look back. I
20 don't recall off the top of my head.

21 **Q. Do you remember, is it 5 or 10**
22 **or 20?**

23 A. Oh, no. It's a small number.

24 **Q. Is it less than five?**

25 A. I believe so. Regeneron

1 Genetics Center is the main one.

2 Q. Regeneron?

3 A. Genetics Center.

4 Q. Genetics Center.

5 And what do they make?

6 A. I don't know all the products
7 that Regeneron makes, but the -- it's
8 specifically the Genetics Center within
9 Regeneron.

10 Q. Okay. Do you have stock in
11 Regeneron?

12 A. I do not.

13 Q. Do you have options?

14 A. I do not.

15 Q. Do you get paid for sitting on
16 the board?

17 A. I had in the past, but I don't
18 now, but I'm not -- again, a scientific
19 advisor, not on a board.

20 Q. Okay. What other companies are
21 you either on the board or a scientific
22 advisor for?

23 A. Prime Medicine.

24 Q. Prime?

25 A. Prime Medicine.

1 **Q. And what does Prime Medicine**
2 **do?**

3 A. They have a technology for gene
4 editing.

5 **Q. And are you on the scientific**
6 **advisory board?**

7 A. The board of directors.

8 **Q. You're on the board of**
9 **directors for Prime Medicine.**

10 **And is that a paid position?**

11 A. It is.

12 **Q. Do you own stock in Prime**
13 **Medicine?**

14 A. I have options, but I don't own
15 stock.

16 **Q. Okay. Were the options issued**
17 **in exchange for you serving on the board?**

18 A. As part of being a member of
19 the board of director, I was given options.

20 **Q. Is that a publicly traded**
21 **company?**

22 A. It is.

23 **Q. Okay. And how long have you**
24 **been on the board of Prime Medicine?**

25 A. I believe a little less than

1 two years.

2 Q. How did you get on -- how did
3 that come about? How did you get approached
4 to be on the board of Prime Medicine?

5 A. I was approached by a firm
6 that's -- places members of boards of
7 directors.

8 Q. They approached you?

9 A. They approached me.

10 Q. Okay. And what does Prime
11 Medicine -- what do they do? What do they
12 make? How do they make money?

13 MS. BROWN: Objection to the
14 form.

15 You can answer, if you know.

16 THE WITNESS: Right now they
17 don't have a product, but they are
18 trying to develop therapies for
19 genetic conditions and for diseases
20 using that prime editing technology.

21 QUESTIONS BY MR. TRACEY:

22 Q. Okay. And the hope is that one
23 day they will have that technology available
24 commercially?

25 MS. BROWN: Objection to the

1 form.

2 THE WITNESS: The hope is that
3 one day they will be able to apply
4 that technology for diseases.

5 QUESTIONS BY MR. TRACEY:

6 Q. And to be used commercially, to
7 sell?

8 A. Yes, correct.

9 Q. Yeah.
10 It's a for-profit company?

11 A. It is a publicly traded,
12 for-profit company.

13 Q. And do your options -- so do
14 you know when your options expire?

15 A. I don't recall.

16 Q. Okay. Any other companies that
17 you are on the boards of?

18 A. Rallybio.

19 Q. Reilly?

20 A. Rallybio, R-a-l-l-y, Bio.

21 Q. And where is Rallybio?

22 A. In Connecticut.

23 Q. And what do they do or make?

24 A. They don't have a commercial
25 product at this time, but, again, they focus

1 on rare diseases and have several things that
2 they're working on.

3 Q. And are you on the board of
4 directors for that company?

5 A. Correct.

6 Q. And is that a paid position?

7 A. Yes, it is compensated.

8 Q. And so both Prime Medicine and
9 Rallybio are positions where you sit on the
10 boards of these companies and you're paid for
11 that?

12 A. That is correct.

13 Q. Now, you told me that with
14 Prime Medicine you also have options.

15 Do you have options with
16 Rallybio?

17 A. I do.

18 Q. Okay. And have you exercised
19 any of those options?

20 A. I have not.

21 Q. Do you have any stock in the
22 company independent of the options?

23 A. I do not.

24 Q. Okay. You told me that company
25 isn't selling a product yet, but it hopes to?

1 A. That is correct.

2 Q. And what type of company is
3 Rallybio? What are they hoping to sell one
4 day?

5 A. They're a pharmaceutical --
6 they're developing treatments for human
7 conditions.

8 Q. What human conditions, if you
9 know?

10 A. So they have things in early
11 stages, some of which are not publicly
12 disclosed.

13 Q. Okay. Are they a -- for
14 autism?

15 A. No, they are not.

16 Q. Are they for any
17 neurodevelopmental disorders?

18 A. They are not.

19 Q. How did you get to be on the
20 board of Rallybio?

21 A. I was approached by someone who
22 tries to recruit boards of directors.

23 Q. Okay. You told me that, didn't
24 you?

25 A. (Witness nods head.)

1 Q. Yeah.

2 And what about Prime Meridian
3 {sic}, how did you get on their board?

4 A. Same process.

5 Q. Oh, it was the same process.

6 Okay. Okay. Any other
7 companies?

8 A. No.

9 Q. So you are -- are you on the
10 scientific advisory board for Regeneron?

11 A. For the Regeneron Genetics
12 Center.

13 Q. But -- okay. But you're not on
14 the board of directors for that company?

15 A. I am not.

16 Q. Okay. Do you know anybody at
17 Johnson & Johnson?

18 MS. BROWN: Objection to the
19 form.

20 You can answer, if you
21 understand.

22 THE WITNESS: I'm sure there's
23 someone at Johnson & Johnson that I
24 know, just because it's a large
25 company with subsidiaries.

1 But off the top of my head,
2 there's no one that I'm working with
3 at Johnson & Johnson that comes to
4 mind.

5 QUESTIONS BY MR. TRACEY:

6 Q. Well, whether you're working
7 with them or not, can you identify a Johnson
8 & Johnson employee that you know?

9 A. No, because I'm not actively
10 working with Johnson & Johnson.

11 Q. What does that mean? What does
12 that have to do with my question?

13 Do you know anybody at Johnson
14 & Johnson?

15 MS. BROWN: I object to the
16 form. She answered the question.

17 THE WITNESS: I can't think of
18 anyone that I know works at Johnson &
19 Johnson. On the other hand, I have a
20 large circle of people that I know,
21 and they move in positions.

22 And I can't say for sure that
23 someone might not be at Johnson &
24 Johnson currently, and I wouldn't have
25 realized that they've moved to Johnson

1 & Johnson.

2 QUESTIONS BY MR. TRACEY:

3 **Q. I see what you're saying.**

4 **Okay. I understand now. Thank you.**

5 **Do you know what the JAKE**
6 **program is at Johnson & Johnson, J-A-K-E?**

7 MS. BROWN: Objection to form.

8 THE WITNESS: I believe there
9 is an app that's used at Janssen.

10 QUESTIONS BY MR. TRACEY:

11 **Q. Good point. Janssen, not J&J.**

12 A. And I believe that's what
13 you're referring to in terms of JAKE.

14 **Q. Yes, that is.**

15 **Are you familiar with the app?**

16 A. I have looked at the app
17 several years ago.

18 **Q. Why did you look at the app**
19 **several years ago?**

20 A. To understand whether or not it
21 would be useful to look at behaviors with
22 autism and to use it for outcome measures to
23 be able to continuously assess those
24 behaviors.

25 **Q. How did you get access to the**

1 **app?**

2 A. I didn't have access, per se.
3 I saw a demonstration of the app.

4 **Q. Oh, where did you see a**
5 **demonstration?**

6 A. I believe I saw it in a
7 demonstration in New Jersey.

8 **Q. Where in New Jersey?**

9 A. At Janssen.

10 **Q. At the Janssen offices?**

11 A. Yes.

12 **Q. Do you remember who presented**
13 **the --**

14 A. It was a group of people.

15 **Q. From Janssen?**

16 A. Correct.

17 **Q. Do you remember any names?**

18 A. I don't.

19 **Q. Okay. Is that the last time**
20 **you've had any interaction with anybody at**
21 **Janssen that you know of?**

22 A. So there is a collaborative
23 from Foundation NIH, or FNIH, that sponsors
24 something called ABC-CT, which is looking at
25 autism biomarkers, and I have been at

1 meetings where Janssen has had
2 representatives there.

3 **Q. And do you remember any of**
4 **their names?**

5 A. I don't know off the top of my
6 head.

7 **Q. Okay. How did you know they**
8 **were from Janssen?**

9 A. It said it in the materials,
10 that they were labeled as Janssen.

11 **Q. Okay. Are you -- was the --**
12 **was the JAKE app something that you were**
13 **interested in using?**

14 A. We're always assessing
15 different ways of measuring behavior, so we
16 were assessing it. We didn't -- I haven't
17 used it personally.

18 **Q. Why not?**

19 A. We haven't moved forward. At
20 the time, this was when I was in a position
21 as director of clinical research at the
22 Simons Foundation, and we hadn't moved
23 forward with studies that would use JAKE app
24 or something like that in person.

25 We just didn't have reason to

1 be able to use anything like that.

2 Q. Do you remember if the JAKE app
3 assessed environmental exposures in pregnant
4 women?

5 A. To my knowledge, I don't recall
6 that it did.

7 Q. All right. Do you have any
8 materials from that JAKE meeting that you
9 attended at Janssen?

10 A. I do not.

11 Q. Did they give you any
12 materials?

13 A. I don't recall.

14 Q. Was there a PowerPoint?

15 A. I'm sure they showed a
16 PowerPoint on the screen.

17 Q. Okay. How long did that
18 meeting last?

19 A. I'm guesstimating, two hours.

20 Q. Okay. Did you go by yourself?

21 A. I don't recall. I think it's
22 likely that someone else from the Simons
23 Foundation was with me, but I don't recall.

24 Q. You mentioned the Simons
25 Foundation a few times.

1 **What is that?**

2 A. The Simons Foundation is a
3 non-profit based in New York City.

4 **Q. You used to be in New York**
5 **City, right?**

6 A. That's correct.

7 **Q. Were you at Columbia?**

8 A. I was.

9 **Q. How many years were you at**
10 **Columbia?**

11 A. I began in 1998 and went
12 through 2023. So I guess 25 years.

13 **Q. Did you enjoy your time at**
14 **Columbia?**

15 A. I did.

16 **Q. Is that a fine institution?**

17 A. It is.

18 **Q. One of the best in the country?**

19 A. I do think Columbia is a very
20 good institution.

21 **Q. Great researchers?**

22 A. Many researchers at Columbia
23 are good.

24 **Q. Yeah.**

25 **Okay. How did you end up at**

1 **Janssen for this two-hour presentation on**
2 **JAKE?**

3 A. We were serving different ways
4 of being able to assess behaviors and
5 understand whether they would be useful for
6 clinical trials and being able to measure
7 changes in behavior over time. So we were
8 investigating many technologies.

9 Q. Okay. So fair enough.

10 I'm trying to figure out how
11 you got -- did you get an invitation? Did
12 you call them? How did that all come about
13 that you ended up at Janssen's offices for
14 two hours in New Jersey?

15 A. I don't recall exactly, but I'm
16 sure that we had heard about it at an autism
17 meeting.

18 Q. Okay.

19 A. And mutually had decided it
20 would be useful to have a demonstration of
21 the app.

22 Q. Okay. Other than that
23 collaborative you told me about -- do you
24 participate in any groups or with any Johnson
25 & Johnson or Janssen or Kenvue employees, any

1 **professional organizations or groups?**

2 A. Can you clarify what you mean
3 by that?

4 Q. I'm not sure.

5 Do you belong to any
6 **professional organizations that also have**
7 **Johnson & Johnson or Kenvue or Janssen**
8 **employees in it?**

9 A. I'm a member of many
10 professional organizations, and I'm sure that
11 there must be some employees that are members
12 of the same organizations, but I don't know
13 who, and I don't know which ones.

14 Q. Okay. Do drug companies
15 **sometimes show up at the professional**
16 **meetings that you attend?**

17 MS. BROWN: Objection to the
18 form.

19 THE WITNESS: Many of the
20 scientific meetings that I attend have
21 good scientists and clinicians that
22 attend, and we have individuals from
23 all different parts of science who
24 attend. So, yes, I believe there
25 probably are people from commercial

1 entities.

2 QUESTIONS BY MR. TRACEY:

3 Q. Well, you know there are,
4 right, Doctor?

5 A. I believe there are. They're
6 part of our scientific community.

7 Q. Sure.

8 You've seen them and talked to
9 them at these meetings, these scientists that
10 work for drug companies, right?

11 MS. BROWN: Objection to the
12 form.

13 THE WITNESS: Again, I talk to
14 scientists at scientific meetings, and
15 many of them are at academic
16 institutions, many of them are part of
17 the government, many of them are part
18 of commercial entities.

19 QUESTIONS BY MR. TRACEY:

20 Q. Right.

21 And the commercial entity part
22 are drug companies, pharmaceutical companies,
23 right?

24 MS. BROWN: Objection to the
25 form.

1 THE WITNESS: In terms of
2 commercial entities, there are many
3 commercial entities in the space where
4 I work in genetics, some of which
5 represent pharmaceutical companies,
6 but there are also other commercial
7 entities that participate in science
8 besides pharmaceutical companies.

9 QUESTIONS BY MR. TRACEY:

10 Q. So there's a whole host of
11 commercial entities, that is, companies, that
12 are trying to make a profit in your space
13 that come to these meetings?

14 MS. BROWN: Objection to the
15 form.

16 THE WITNESS: So in the
17 meetings that I attend, there are
18 scientists in many different parts of
19 the world represented in many sectors
20 of the -- of society. And --

21 QUESTIONS BY MR. TRACEY:

22 Q. Do you remember my question?

23 MS. BROWN: Well, she wasn't
24 done answering it, so let's let her do
25 that.

1 THE WITNESS: And within this,
2 many different scientists are able to
3 contribute in many different ways.
4 And in many cases, where we've made
5 progress in terms of public health,
6 has been through being able to have
7 collaborations and partnerships that
8 go across many of these different
9 sectors.

10 QUESTIONS BY MR. TRACEY:

11 Q. Collaborations and partnerships
12 with these many sectors including drug and
13 pharmaceutical companies?

14 MS. BROWN: Objection. Vague.

15 THE WITNESS: Again, what has
16 always been, at least in the way I
17 have collaborated, has been
18 collaborations between good
19 scientists, good doctors, people who
20 are qualified to do whatever the
21 objective is. And in some cases, they
22 have been part of commercial
23 companies, in some cases they have
24 been part of pharmaceutical companies,
25 but they have always been good

1 scientists doing good science.

2 MR. TRACEY: I'm going to

3 object to nonresponsive.

4 QUESTIONS BY MR. TRACEY:

5 Q. Ma'am, do you know what it is
6 when a lawyer says an answer is
7 nonresponsive?

8 MS. BROWN: No. Objection.

9 Don't -- don't --

10 QUESTIONS BY MR. TRACEY:

11 Q. I think you do. Don't you?

12 A. I apologize --

13 MS. BROWN: It's argumentative,
14 and it's irrelevant.

15 THE WITNESS: I apologize, I
16 thought I did answer the question. If
17 you would like to ask the question
18 again, I will try again to be clearer
19 in my answer.

20 QUESTIONS BY MR. TRACEY:

21 Q. You collaborate at these
22 meetings with employees from pharmaceutical
23 companies?

24 MS. BROWN: I object --

25 MR. TRACEY: That was my

1 question.

2 MS. BROWN: It's been asked and
3 answered. I object.

4 THE WITNESS: Again, as we have
5 discussions, scientific discussions,
6 there are scientists in many different
7 sectors who can speak and who can work
8 together. In some cases, those
9 include individuals who are in
10 pharmaceutical companies or other
11 commercial enterprises who will
12 collaborate with good scientists in
13 other locations, including academia.

14 QUESTIONS BY MR. TRACEY:

15 **Q. How often do these good**
16 **clinicians from commercial enterprises take**
17 **place with you during the year?**

18 MS. BROWN: Objection. Vague.

19 THE WITNESS: Can you rephrase
20 the question to understand how you
21 want the accounting to occur? When
22 you say how many times, how should I
23 think about that?

24 QUESTIONS BY MR. TRACEY:

25 **Q. I'm not sure. You said that**

1 **many times you collaborate with these**
2 **commercial enterprises at these scientific**
3 **meetings.**

4 **Does that happen once a year?**
5 **Ten times a year?**

6 A. So if I understand your
7 question, I probably attend and/or present
8 at, say, ten scientific meetings per year.
9 And I haven't done an inventory of the entire
10 attendee list, but I wouldn't be surprised if
11 there's at least one person from a
12 pharmaceutical company at all of those
13 meetings.

14 **Q. Okay. When's the last time --**
15 **what was the last meeting that you presented**
16 **at?**

17 A. Let me think about this.
18 There was a scientific meeting
19 in Taiwan on newborn screening. I believe
20 that was the last one that I presented on.

21 **Q. And do you remember**
22 **approximately when that was?**

23 A. Over the summer.

24 **Q. Do you remember who sponsored**
25 **that meeting?**

1 A. I don't.

2 **Q. Is it your experience that many**
3 **times these meetings are sponsored by drug or**
4 **pharmaceutical companies?**

5 MS. BROWN: Objection to the
6 form.

7 THE WITNESS: There can be at
8 some scientific meetings booths where
9 a pharmaceutical company will have a
10 booth, and in that way, they pay for
11 the booth.

12 QUESTIONS BY MR. TRACEY:

13 **Q. Yeah.**

14 **And at these -- I've been to**
15 **these meetings, some of these, and there will**
16 **be -- pharmaceutical companies will have**
17 **booths that are manned with materials and**
18 **people so that if at these meetings you want**
19 **to talk about whatever it is they're selling,**
20 **you can do that?**

21 MS. BROWN: I object.

22 Is that a question?

23 MR. TRACEY: It is. Most of my
24 statements are going to be in --
25 interrogative.

1 MS. BROWN: Yeah, this looked
2 like it was a declarative statement.
3 So I'll object.

4 MR. TRACEY: Well, when my
5 voice rises at the end, that's
6 typically a cue that it's a question.

7 MS. BROWN: Okay. It looks
8 like a statement. I object.

9 THE WITNESS: So at many
10 scientific meetings, there are booths
11 where individuals are there to be able
12 to ask questions that attendees at the
13 meetings may approach them about.

14 QUESTIONS BY MR. TRACEY:

15 Q. And they'll have stacks of
16 material, oftentimes, that they hand out to
17 attendees at these meetings, right?

18 MS. BROWN: Objection to the
19 form.

20 THE WITNESS: For individuals
21 asking questions, they often have
22 materials to be able to answer their
23 questions.

24 QUESTIONS BY MR. TRACEY:

25 Q. And then sometimes in these

1 **meetings, you'll see the literature or the**
2 **promotional materials will say, sponsored by**
3 **Johnson & Johnson or Prime Medicine, or it's**
4 **whoever it is, right?**

5 MS. BROWN: Same objection.

6 THE WITNESS: Depending on how
7 the meeting is set up, there may be
8 sponsors that would be, for instance,
9 on a study web -- or rather, a meeting
10 website, yes, if they've sponsored the
11 meeting.

12 QUESTIONS BY MR. TRACEY:

13 **Q. Has a pharmaceutical company**
14 **ever sponsored any of your research or help**
15 **pay for it?**

16 A. Yes, they have.

17 **Q. How many times?**

18 A. Three times that they've
19 sponsored my research, and there have been
20 times where I've been part of a clinical
21 trial as an individual site amongst many
22 sites in the clinical trial.

23 **Q. How many times -- so three**
24 **times they've sponsored your research.**

25 Do you recall the last time

1 **your research was sponsored by a drug**
2 **company?**

3 A. Currently, two of my studies
4 are sponsored.

5 Q. Oh.
6 **What drug companies?**

7 A. Ovid and Sanofi.

8 Q. **Sanofi?**

9 A. Sanofi.

10 Q. **And -- sorry.**

11 A. Ovid. O-v-i- --

12 Q. **I told you I'm hard of hearing.**

13 A. I'm sorry. O-v-i-d.

14 Q. Okay. And then you said -- so
15 those are two current studies being sponsored
16 by drug companies?

17 A. That is correct.

18 Q. And how many in the past
19 have -- how many drug companies in the
20 past -- or how many studies of yours in the
21 past have been sponsored or paid for by drug
22 companies?

23 A. I've had one additional sponsor
24 for one additional study.

25 Q. **Who was that?**

1 A. Biogen.

2 Q. Biogen.

3 Okay. And then you said you've
4 been part of collaborations for clinical
5 research that were sponsored or paid for by
6 drug companies?

7 A. Let me clarify.

8 Q. Okay.

9 A. I've been part of clinical
10 trials, and the clinical trial has been
11 sponsored by the company testing a potential
12 therapy.

13 Q. And how many clinical trials
14 have you been a part of?

15 A. We've been part of one by
16 Rhythm.

17 Q. Rhythm is the company?

18 A. Rhythm is the company.

19 Q. Okay. Any others?

20 A. No.

21 Q. Pardon me?

22 A. No.

23 Q. No.

24 Okay. When you were at
25 Columbia, would drug company employees come

1 **see you from time to time?**

2 MS. BROWN: Objection to the
3 form.

4 Go ahead.

5 THE WITNESS: Drug companies
6 would not come to me at Columbia, no.

7 QUESTIONS BY MR. TRACEY:

8 **Q. Well, would they approach you**
9 **either by e-mail or phone from time to time?**

10 A. If people approached me, per
11 se, it was usually at a scientific meeting
12 where many of us were together and where they
13 would present to a large number of us as an
14 audience.

15 **Q. Did you ever have meetings that**
16 **were preplanned at these scientific**
17 **conferences with drug companies?**

18 A. Occasionally, there would be an
19 educational session, and you were free to
20 sign up and to be able to attend the session.

21 **Q. Okay. You wrote a report in**
22 **this case, right?**

23 A. I did.

24 **Q. It's in front of you?**

25 A. It is.

1 **Q. I've forgotten the exhibit**
2 **number.**

3 **Can you tell me what it is?**
4 **You're so far way.**

5 **A. 305.**

6 **(Chung Exhibit 305 marked for**
7 **identification.)**

8 **QUESTIONS BY MR. TRACEY:**

9 **Q. 305.**

10 **Well, let me ask you this**
11 **before I get into your report.**

12 **Have you ever had an occasion**
13 **outside of litigation to assess the**
14 **scientific literature on a subject and come**
15 **to conclusions about what the data says?**

16 **A. Yes, I have.**

17 **Q. That's something you do fairly**
18 **regularly?**

19 **A. That's a routine part of what I**
20 **do as a scientist.**

21 **Q. And are there methods or**
22 **frameworks that you employ or utilize to**
23 **assess the data so that you can come to**
24 **conclusions about what the data means to you?**

25 **A. Yes. As a scientist, I have**

1 many criteria and ways of being able to
2 approach a paper, a manuscript, a set of
3 data, to assess the validity and how
4 rigorously the study has been performed.

5 Q. Do those -- what do we call
6 those? Are those frameworks? Are they --
7 are they methodologies? What do we call that
8 when you do that in your
9 outside-of-litigation work?

10 A. I would say it's how I assess
11 the -- well, any dataset, how I assess the
12 scientific data.

13 Q. One paper you published, I saw
14 recently, you actually suggested what was
15 called a framework for evaluating scientific
16 evidence.

17 Is that a term you're familiar
18 with?

19 A. I'm familiar with the term.
20 I'm not sure which paper or which manuscript
21 you're referring to.

22 Q. Okay. Maybe we'll talk about
23 it later.

24 I'm just trying to understand,
25 is there a term or a methodology that you

1 **employ outside of litigation when you're**
2 **assessing scientific literature?**

3 A. Scientific literature is a
4 pretty broad term, and it refers to many
5 different data types in doing this. So it's
6 hard to be able to say that there's one
7 single way that we do this.

8 But, in general, we try and use
9 the methods that are available at the time
10 when the data are available to assess the
11 validity, the rigor with which the study was
12 performed, the reproducibility, and I'd say
13 those are general things that we do
14 throughout science.

15 **Q. Okay. Are you aware that there**
16 **are methodologies that are employed by**
17 **scientists to assess scientific literature**
18 **and data and then reach conclusions that have**
19 **names?**

20 MS. BROWN: Objection to the
21 form.

22 THE WITNESS: Again, science is
23 a broad field, but, yes, I'm sure
24 fields of science have particular
25 methods that they employ.

1 QUESTIONS BY MR. TRACEY:

2 Q. For example, are you familiar
3 with something called a weight of the
4 evidence methodology to assess scientific
5 evidence?

6 A. I'm not personally familiar
7 with that method.

8 Q. Okay. And I saw in another
9 deposition somebody asked you if you employed
10 the Bradford Hill criteria in assessing
11 causation in a case, and you seemed
12 unfamiliar with that.

13 Is that true?

14 MS. BROWN: Objection to the
15 form.

16 THE WITNESS: I'm familiar with
17 the criteria. It's not something that
18 as a geneticist applies to a lot of
19 the genetic research that I do, but
20 I'm familiar with the criteria and
21 familiar with others who use the
22 criteria.

23 QUESTIONS BY MR. TRACEY:

24 Q. Have you ever employed the
25 Bradford Hill criteria in assessing

1 **causation, ever?**

2 A. I think there's some of the
3 Bradford criteria that are useful across
4 science in terms of looking at
5 reproducibility, looking at order of
6 magnitude in terms of the effect size that we
7 see, in terms of assessing plausibility. I
8 think those are general sort of criteria that
9 are used scientifically across multiple
10 methods, my own field included.

11 **Q. So you use some of the criteria**
12 **of Bradford Hill?**

13 A. Let me try and rephrase that.
14 Some of the Bradford Hill
15 criteria are the same way that we approach
16 science in a more generic and general way.
17 And so I think those are, in terms of what I
18 use in my routine practice and my routine
19 research, things that I routinely use as I
20 assess scientific data.

21 **Q. Have you ever used the Bradford**
22 **Hill criteria and employed it methodically to**
23 **assess scientific evidence?**

24 A. I'm not an epidemiologist. I'm
25 a geneticist.

1 **Q. Good point.**

2 A. And as I do that, I tend to use
3 the methods that I personally use in my field
4 in terms of genetics. And to the extent that
5 some of those criteria are overlapping, I'd
6 say that's routine in my practice in terms of
7 assessing the scientific literature.

8 **Q. I'm not sure what the answer**
9 **is.**

10 MS. BROWN: Objection.

11 QUESTIONS BY MR. TRACEY:

12 **Q. Do you use -- have you used the**
13 **Bradford Hill criteria -- have you employed**
14 **the criteria as set forth by Bradford Hill to**
15 **assess scientific -- to assess causation?**

16 MS. BROWN: Objection. Asked
17 and answered.

18 THE WITNESS: So from a formal
19 point of view, I have not
20 systematically gone through the
21 Bradford Hill criteria.

22 QUESTIONS BY MR. TRACEY:

23 **Q. Okay.**

24 A. But on the other hand, many of
25 the Bradford Hill criteria -- in fact, I

1 would say that the main drivers in terms of
2 an order of priority and importance are the
3 same and overlapping features that we use to
4 assess scientific data in the fields in which
5 I am an expert.

6 (Chung Exhibit 348A marked for
7 identification.)

8 QUESTIONS BY MR. TRACEY:

9 Q. Okay. Let me hand you
10 Exhibit 348. Is it A? I should have looked.
11 348A.

12 It's an excerpt from a
13 deposition you gave a few years ago where you
14 were asked about the Bradford Hill criteria.

15 MS. BROWN: Counsel, do you
16 have the entire deposition transcript
17 available?

18 MR. TRACEY: No.

19 MS. BROWN: If you have it, can
20 you e-mail it, at least? Does
21 somebody have it?

22 MR. TRACEY: Somebody probably
23 has it.

24 MS. BROWN: Okay.

25 MR. TRACEY: Sure. Let me --

1 yeah, I assume. It's her testimony.

2 MS. BROWN: Okay. I'm just
3 going to object to the extent she
4 needs to look at more of this half a
5 page we have printed out.

6 But if you have the whole thing
7 available, it would be great if she
8 could take a look at it.

9 MR. TRACEY: Okay. Yeah.
10 Well, I think it's two pages, not half
11 a page.

12 QUESTIONS BY MR. TRACEY:

13 Q. And so on page 221 -- oh,
14 **sorry. Let's see if it reduced it.**

15 This is in the case we were
16 **talking about earlier. This is the case**
17 **Nichole Daniels-Feasel versus Forest**
18 **Pharmaceuticals.**

19 You were an expert in that
20 **case.**

21 A. That is correct.

22 Q. And on page 221 of your
23 **deposition, you were asked:**

24 **"Have you applied the Bradford**
25 **Hill criteria to your genetic theory of**

1 autism?"

2 Do you see that?

3 A. Yes, I do.

4 Q. And your answer was:

5 "Again, I'm not someone who
6 thinks about Bradford Hill in terms of
7 doing this.

8 "I think about genetics as a
9 discipline, as a scientific area."

10 Then you were asked:

11 "So you did not apply Bradford
12 Hill, right?"

13 And you say:

14 "Again, I don't think in terms
15 of Bradford Hill."

16 Is that -- was that your
17 testimony?

18 A. That's what's in this printout,
19 yes.

20 Q. Well, that was true, wasn't it?

21 A. I believe that's consistent
22 with what I just stated in terms of the way I
23 approach things scientifically as a
24 geneticist.

25 Q. Okay. So you do not think in

1 **terms of Bradford Hill?**

2 A. Again, as I think -- I think --
3 I am a geneticist, and I think as a
4 geneticist. That's my discipline. That's my
5 field of expertise.

6 As we think about this, there
7 are many of the same scientific criteria that
8 overlap between Bradford Hill criteria and
9 how I think about science and how I think
10 about data.

11 And those are the criteria that
12 I tend to use, and those also happen to be
13 major drivers in terms of causation under
14 Bradford Hill as I understand those criteria.

15 **Q. You don't -- in your report you**
16 **don't mention Bradford Hill one time, do you?**

17 A. I could do a search, but I
18 don't recall that I did mention Bradford
19 Hill, per se.

20 **Q. I don't remember seeing a**
21 **weight of the evidence methodology employed**
22 **in your report either.**

23 **Do you remember that?**

24 A. I'm sure I did not state using
25 a weight of the evidence.

1 **Q. Did you tell us in your report**
2 **what methodology you were employing to**
3 **evaluate the scientific evidence and reach**
4 **whatever conclusions you reached?**

5 A. I used the -- I wouldn't say
6 that it's got a name in terms of the way that
7 we approach data, but we do think of
8 geneticists as being able to look at the
9 data.

10 And in particular because
11 autism and ADHD are so heavily genetically
12 determined, my opinion is largely in terms of
13 trying to understand the role of genetics and
14 as we think about how other things are
15 brought into play, whether those can be
16 explained by genetic factors.

17 **Q. Did you -- did you reference**
18 **for the Court or the jury or anybody to see**
19 **the methodology you were employing so that we**
20 **could go look it up ourselves?**

21 A. Again, as geneticists, there
22 are many, many different methods that we use.
23 I don't know that there's one handbook or one
24 name that we use because there's so many
25 types of analyses that are performed.

1 But as geneticists, we tend to
2 be extremely data-driven, and with that, very
3 rigorous in terms of what we will accept in
4 terms of causation, and we -- I applied those
5 same methods to the literature that I
6 reviewed.

7 **Q. What methods?**

8 A. Being able to assess genetic
9 contributions to conditions, being able to
10 understand the relative weight of the
11 contributions and how to design studies to
12 assess genetic contributions to conditions.

13 **Q. What weight did you assign the**
14 **evidence, and where in your report can I find**
15 **the assignment of the weight for the evidence**
16 **that you evaluated?**

17 A. As an example, when one looks
18 at heritability estimates for conditions, one
19 can get an assessment of the variance for
20 that phenotype, how much of that is genetic
21 and for both of the conditions of autism and
22 ADHD.

23 Those estimates are extremely
24 high, 80 to 90 percent; in other words --

25 **Q. Whoa, whoa, whoa, whoa.**

1 MS. BROWN: She's got to finish
2 that. No, no, no, no.

3 MR. TRACEY: I'm not asking
4 about that yet.

5 MS. BROWN: No, hold on.

6 QUESTIONS BY MR. TRACEY:

7 Q. I'm trying to figure out,
8 Dr. Chung --

9 MS. BROWN: Sir? Sir? She's
10 not done with her answer. She's got
11 to finish her answer, and then you can
12 follow up.

13 Please finish your answer, and
14 then if you want to follow-up, that's
15 perfectly fine.

16 Go ahead.

17 THE WITNESS: So for both of
18 those conditions, autism and ADHD,
19 being 80 to 90 percent of the variants
20 attributable to genetic factors, that
21 is the overwhelming contributor to
22 those conditions.

23 And everything else has to be
24 looked at within the context of
25 genetics and understand whether

1 genetics has been accounted for in
2 terms of compounds.

3 QUESTIONS BY MR. TRACEY:

4 **Q. Do you remember my question?**

5 A. If you don't mind repeating it,
6 I'd appreciate it.

7 **Q. Do you not remember it?**

8 A. Just to make sure I've got it
9 right, I would appreciate it if you'd repeat
10 it.

11 **Q. What do you think you were**
12 **answering?**

13 MS. BROWN: Objection.
14 Argumentative.

15 She's asked for clarification.
16 Our effort here is to get truthful and
17 accurate testimony.

18 QUESTIONS BY MR. TRACEY:

19 **Q. Where can I find a description**
20 **of the methodology you employed in your**
21 **report?**

22 MS. BROWN: I object. Asked
23 and answered.

24 QUESTIONS BY MR. TRACEY:

25 **Q. Just pull out your report and**

1 point it out to me. I don't want to have to
2 guess.

3 A. As scientists, we don't have --
4 because there are so many different methods
5 that are applied, there is not one single
6 method that we use to apply to all of the
7 literature and all of the scientific methods
8 that are so rapidly evolving.

9 Q. Let me ask you this. Where do
10 I find the search terms that you employed to
11 actually identify the relevant literature?

12 A. So can you repeat the question,
13 please?

14 Q. Yes.

15 Where in your report can I turn
16 to and find the search terms that you
17 employed to identify the relevant literature
18 in this case?

19 A. I don't believe in my report I
20 list search terms.

21 Q. You certainly do that in your
22 academic life, don't you?

23 A. I do scientific searches, that
24 is true, in my -- in my academic life.

25 Q. And you identify what search

1 **terms you're using to identify relevant**
2 **literature, don't you?**

3 A. Depending on the question,
4 there may be a very simple question in which
5 one can do a simple literature review in
6 terms of being able to use search terms.

7 The methods to understand a
8 complex body of data is not simply a matter,
9 however, of a literature search.

10 **Q. Well, you want to be**
11 **transparent, Dr. Chung, don't you?**

12 We want the judge and the jury
13 to understand how you reached the conclusions
14 you reached.

15 **Right?**

16 MS. BROWN: Objection to the
17 form of the question.

18 THE WITNESS: I believe in
19 transparency.

20 QUESTIONS BY MR. TRACEY:

21 **Q. Transparency is fundamental to**
22 **the scientific method, isn't it?**

23 A. I believe in transparency. I
24 believe in being able to, as a scientific
25 community, share, to be able to arrive at the

1 most accurate information.

2 And by sharing that, we evolve
3 our thinking over time and continue to
4 improve in the accuracy of that information.

5 **Q. And what you didn't share in**
6 **this case is the search term strategy you**
7 **employed to identify literature, right?**

8 A. I didn't --

9 MS. BROWN: Objection to the
10 form.

11 Go ahead.

12 THE WITNESS: I did not
13 specifically list search terms, in
14 large part, because many of my
15 colleagues -- this would have been
16 intuitive.

17 QUESTIONS BY MR. TRACEY:

18 **Q. It would have been intuitive to**
19 **who?**

20 A. To colleagues such as myself.

21 **Q. I see.**

22 **So you didn't feel the need to**
23 **share the search terms with the Court because**
24 **you think it would be intuitive to colleagues**
25 **like yourself?**

1 MS. BROWN: Objection to the
2 form. Argumentative. Misstates her
3 testimony.

4 THE WITNESS: In terms of the
5 review that I did, I do believe that I
6 did a comprehensive analysis for the
7 specific questions in terms of
8 associations between acetaminophen,
9 autism and ADHD.

10 QUESTIONS BY MR. TRACEY:

11 Q. No doubt. I don't doubt you
12 believe it, ma'am, but we were talking about
13 sharing data and transparency, right?

14 A. I believe we were talking about
15 search terms. There wasn't anything -- I
16 think anything cloaked in terms of any
17 secrecy for that.

18 I simply thought it was obvious
19 in terms of the question, that that's how I
20 would search, and that's how, even beyond
21 another scientist intuitively, one would have
22 searched on those terms.

23 Q. Okay. What was the question
24 you were answering? That's actually not in
25 your report either, is it?

1 MS. BROWN: Objection to the
2 form.

3 QUESTIONS BY MR. TRACEY:

4 Q. Is the question that you were
5 answering in your report for -- that you
6 identify that for the Court so we knew what
7 question that you were answering in your
8 report.

9 MS. BROWN: Objection to the
10 form.

11 THE WITNESS: Give me one
12 second --

13 QUESTIONS BY MR. TRACEY:

14 Q. Yeah, please, by all means.

15 A. -- to see specifically how I
16 stated this in my report.

17 So in my report, I have several
18 opinions that are listed, and within that,
19 most individuals would have thought to look
20 at the terms used within the opinions that I
21 stated and doing searches on those keywords.

22 And that was, in fact, what I
23 have done.

24 Q. Okay. But what was the --
25 where is the question in your report that you

1 **were answering for the Court?**

2 A. I'm rendering several opinions
3 that are listed under point number 3 and that
4 go on for a couple of pages thereafter.

5 **Q. What page are you on?**

6 A. Page 2, 3 and into page 4.

7 **Q. And is there a question that**
8 **you're answering on 2, 3 and 4?**

9 A. I'm rendering --

10 **Q. Is there a question identified?**
11 **I should ask a better question.**

12 A. I'm rendering opinions in those
13 cases under points 2 and 3.

14 **Q. You say, "Genetic variants**
15 **account for most known causes of ASD and**
16 **ADHD"?**

17 A. I do say that.

18 **Q. Okay. All right. We're going**
19 **to come back to that.**

20 **But there is no question you've**
21 **identified in your report that you were**
22 **answering that I could find.**

23 **Is that a fair assessment?**

24 MS. BROWN: Objection to the
25 form. Misstates testimony.

1 QUESTIONS BY MR. TRACEY:

2 Q. For example, there's no place
3 in your report where you say, I was asked by
4 Johnson & Johnson to answer the following
5 question?

6 MS. BROWN: Objection to the
7 form.

8 THE WITNESS: Well, number one,
9 I wasn't asked by Johnson & Johnson
10 within any of the report or any of the
11 work that I was thinking through.

12 Point number --

13 QUESTIONS BY MR. TRACEY:

14 Q. Who asked you to do it, ma'am?

15 MS. BROWN: Well, let her
16 finish, please.

17 THE WITNESS: Point number two,
18 within my report on page 2 I believe
19 is what you're getting at in terms of
20 the scope of what I was addressing.

21 QUESTIONS BY MR. TRACEY:

22 Q. Where is that?

23 A. Page 2, number 2.

24 Q. Yeah, I'm sorry.

25 Where it says, "This report

1 identifies the robust and rigorously
2 replicated scientific evidence in the
3 published literature demonstrating known
4 genetic ideology about ASD and ADHD"?

5 A. And the following sentence, "My
6 report also addresses the published
7 epidemiological studies on prenatal
8 acetaminophen exposure and outcomes
9 purportedly related to ASD and/or ADHD to
10 assess whether these studies have properly
11 accounted for genetic confounders."

12 Q. In fact, in that last sentence,
13 you address the methodologies of the
14 plaintiffs' experts, don't you?

15 A. I'm sorry, can you clarify?

16 Q. Yeah. That last sentence,
17 finally -- let's put that -- can we have that
18 up on the screen?

19 "Finally, my report addresses
20 the various methodologies and data used by
21 plaintiff experts in their expert reports."

22 That's what you claim to be
23 doing, right -- one of the things you claim
24 to be doing?

25 A. One of the things my report

1 addresses, yes, are some of the plaintiff
2 experts.

3 **Q. The plaintiffs' experts did**
4 **employ methodologies that they identified,**
5 **didn't they?**

6 A. Can you repeat that?

7 **Q. Yes.**

8 **The plaintiffs' expert in this**
9 **case did identify methodologies that they**
10 **employed?**

11 MS. BROWN: Objection to the
12 form.

13 THE WITNESS: Yes, the
14 plaintiffs did identify methods that
15 they employed.

16 QUESTIONS BY MR. TRACEY:

17 **Q. And you critiqued those methods**
18 **in your report, didn't you, or those**
19 **methodologies or the conclusions reached**
20 **based on those methodologies, right?**

21 A. I point out that some of the
22 methods need to be comprehensive in their
23 designs and in particular to be able to
24 include genetics, which accounts for the
25 major contributor to both A -- autism and

1 ADHD.

2 **Q. Okay. And I want to critique**
3 **your methodology, Dr. Chung.**

4 **Is that fair?**

5 MS. BROWN: Objection to the
6 form.

7 THE WITNESS: I'm always open
8 to critique.

9 QUESTIONS BY MR. TRACEY:

10 **Q. Where do I find your**
11 **methodology so that I can critique it in your**
12 **report?**

13 MS. BROWN: I object. That's
14 been asked and answered four times.

15 THE WITNESS: As I've said, the
16 field of genetics and genomics in
17 particular is extremely complex and
18 rapidly evolving. I would argue one
19 of the most rapidly evolving in all of
20 science.

21 Because of that, we tend not to
22 have very rigid sort of rule books in
23 terms of how this is done, so as we do
24 this, I can only point to I use state
25 of the art methods that a geneticist

1 or a genomicist would use in my field.

2 QUESTIONS BY MR. TRACEY:

3 **Q. There's no rigid set of methods**
4 **that are employed in genetics?**

5 A. Again, you've identified a very
6 large field of genetics and genomics. For
7 some things in which we try to come to
8 consensus for a field, yes, there are
9 guidelines.

10 For instance, for ACMG variant
11 interpretation, there are guidelines so there
12 are -- there is consistency. That's --

13 **Q. There sure are, aren't there?**

14 A. That is different --

15 MS. BROWN: Let her finish,
16 Mr. Tracey. You're cutting her off.

17 QUESTIONS BY MR. TRACEY:

18 **Q. I'm sorry, Doctor. I thought**
19 **you were finished.**

20 A. That is --

21 MS. BROWN: Go ahead. Please
22 finish.

23 THE WITNESS: That is
24 different, however, from what I
25 understood your question to be.

1 QUESTIONS BY MR. TRACEY:

2 Q. Okay. But to get back to my
3 original question, there is no, none -- no
4 methodology identified in your report that
5 anybody could critique because you didn't
6 identify one?

7 MS. BROWN: I object. That's
8 been asked and answered and misstates
9 her testimony.

10 THE WITNESS: Again, there is
11 no specific method that I've referred
12 to in my report because in the field,
13 there is no single method to refer to.

14 QUESTIONS BY MR. TRACEY:

15 Q. For example, I can find no
16 place in your report where you assess the
17 literature and you weigh the literature,
18 right?

19 MS. BROWN: Objection to the
20 form.

21 THE WITNESS: Again, I don't
22 reference, nor do I know there to be,
23 a particular weight or mathematical
24 formula that one uses. Again, that is
25 not standard within my field of

1 science.

2 QUESTIONS BY MR. TRACEY:

3 Q. Are you aware of generally
4 accepted scientific methodologies that employ
5 just that technique?

6 MS. BROWN: I object as vague.

7 QUESTIONS BY MR. TRACEY:

8 Q. Where they weigh literature and
9 assign a grade to it, a weight to it?

10 MS. BROWN: Same objection.

11 You can answer, if you
12 understand.

13 THE WITNESS: I don't know all
14 of science. I do know that when it
15 comes to some things with clinical
16 care guidelines, as an example, people
17 will have ratings based on how much
18 evidence there is to support certain
19 claims.

20 QUESTIONS BY MR. TRACEY:

21 Q. Much of the literature that you
22 evaluated in this case was epidemiology,
23 right?

24 A. Some of the literature that I
25 evaluated was epidemiology, in particular

1 with an eye to study design and how genetics
2 was or was not included in the study design
3 and the conclusions that were drawn.

4 Q. And nowhere in your evaluation
5 of the epidemiological literature can I find
6 a weighing of the epidemiological evidence by
7 you, right?

8 MS. BROWN: Objection. Vague.

9 THE WITNESS: I have not
10 employed a weighting factor.

11 QUESTIONS BY MR. TRACEY:

12 Q. Okay. And we've talked about
13 already, we don't know what your search terms
14 were because you didn't identify them, right?

15 MS. BROWN: Objection to the
16 form.

17 THE WITNESS: I believe what I
18 stated is that the search terms were
19 obvious from how I had stated in my
20 report what my report was covering.

21 QUESTIONS BY MR. TRACEY:

22 Q. If I wanted to recreate your
23 search terms, where in your report would I
24 find them obviously?

25 A. Again, within my report on

1 pages 2, 3 and 4, where I outline what my
2 opinions are.

3 **Q. But there's not one search term**
4 **on those pages, Doctor, is there?**

5 A. No, there would not be one
6 search term. There would have been a number
7 of search terms.

8 **Q. I'm presuming that you actually**
9 **did research, right?**

10 A. I did. The search terms, as an
11 example, were "autism," "ADHD,"
12 "acetaminophen," "prenatal" and "genetics."

13 **Q. Okay. That's nowhere in your**
14 **report?**

15 A. It was something that was
16 intuitive and obvious to me based on the
17 scope of my report outlined in my opinions.

18 **Q. What if your research terms**
19 **missed some relevant literature? Is that**
20 **possible?**

21 MS. BROWN: Objection to the
22 form.

23 THE WITNESS: Anything is
24 possible, but I believe that I did a
25 comprehensive search as well as can be

1 done to the standard in my field.

2 QUESTIONS BY MR. TRACEY:

3 Q. When you reviewed the expert
4 reports or the expert rebuttal reports, were
5 you struck that you missed some relevant
6 literature?

7 MS. BROWN: I object to the
8 form of the question.

9 THE WITNESS: I was not struck
10 that I missed relevant literature.

11 QUESTIONS BY MR. TRACEY:

12 Q. Did you add any literature
13 since your report to -- to your report? Did
14 you add any literature?

15 MS. BROWN: I object as vague.

16 THE WITNESS: Any work that I
17 do is an iterative process. Science
18 is an iterative process, and there
19 were multiple revisions to the reports
20 as this evolved over time.

21 QUESTIONS BY MR. TRACEY:

22 Q. So the answer is, yes, you did
23 add literature after your report was filed,
24 right? Some of it I got last night.

25 MS. BROWN: I object.

1 Add to what?

2 QUESTIONS BY MR. TRACEY:

3 Q. Well, add to your materials
4 considered list. The materials you
5 considered.

6 MS. BROWN: I object as vague.

7 Is the question, did she serve
8 a supplemental reliance list?

9 MR. TRACEY: No.

10 MS. BROWN: I object.

11 QUESTIONS BY MR. TRACEY:

12 Q. Let me ask you this.

13 You didn't weigh -- we talked
14 about the fact that you didn't weigh in a way
15 that's -- that I can find in your report the
16 epidemiological literature, right?

17 MS. BROWN: Objection.

18 Misstates testimony.

19 THE WITNESS: Again, we've
20 stated this. I didn't use a weighting
21 factor.

22 QUESTIONS BY MR. TRACEY:

23 Q. Yeah.

24 And you also evaluated
25 preclinical studies, animal studies, right?

1 A. I did not attempt to do a
2 comprehensive review of animal studies.
3 Animals don't have autism. Animals, in terms
4 of their relevance, are not something that I
5 focus on.

6 **Q. Okay. That all may be true,**
7 **but you identified animal studies in your**
8 **report that you reviewed, correct?**

9 A. Again, I did not attempt to do,
10 nor did I do, a comprehensive literature
11 review or assessment of all animal studies
12 that might have been relevant.

13 **Q. Okay. Fair enough.**
14 **So you didn't even attempt to**
15 **do a comprehensive analysis of the animal**
16 **literature?**

17 MS. BROWN: She literally just
18 said that.

19 I object. Asked and answered.

20 THE WITNESS: Again, as I think
21 about a uniquely human condition of
22 autism and what informs causation for
23 autism, I start with the condition. I
24 start with humans. I start with
25 people. And that's the most relevant

1 information.

2 QUESTIONS BY MR. TRACEY:

3 Q. Okay. Can you answer my
4 question?

5 MS. BROWN: Objection. She
6 did.

7 QUESTIONS BY MR. TRACEY:

8 Q. You did not attempt, Dr. Chung,
9 to do a comprehensive search and evaluation
10 of the animal literature, correct?

11 MS. BROWN: Asked and answered.

12 THE WITNESS: I did not attempt
13 to do a comprehensive literature --

14 QUESTIONS BY MR. TRACEY:

15 Q. Okay.

16 A. -- review of the animal
17 studies.

18 Q. And even what you did do,
19 contained in your report, there is no effort
20 made to weigh or grade the animal studies?

21 A. Again, I did not use the term
22 nor a methodology of weighting, per se. I'll
23 again try and explain, and I know this is
24 complicated, so --

25 Q. You don't need to explain,

1 **Doctor.**

2 MS. BROWN: Wait, wait. But
3 she does need to finish. Let her
4 finish.

5 MR. TRACEY: But I just said
6 you don't need to --

7 MS. BROWN: No, no, no. She's
8 not done.

9 MR. TRACEY: I don't need an
10 explanation.

11 MS. BROWN: She's going to give
12 it because you asked a question.
13 Please let her finish, and then you
14 can follow up.

15 Go ahead, Doctor.

16 MR. TRACEY: Okay.

17 THE WITNESS: So what happens
18 in science, especially with rapidly
19 evolving fields, is we continue to
20 iterate in terms of being able to
21 design better and better methods to be
22 able to understand the fundamental
23 question that we're asking.

24 So as we approach this in my
25 field, that idea of weighting is

1 really replaced by what I would call
2 an iterative process of refining both
3 methods to be able to assess the
4 comprehensive models that can be able
5 to understand issues of causation. So
6 I think it's a different field.

7 And I apologize, but I just
8 think we apply different methodologies
9 as a different discipline.

10 QUESTIONS BY MR. TRACEY:

11 **Q. The problem is, you didn't tell**
12 **me what your methodologies are in your report**
13 **for me to critique, right?**

14 MS. BROWN: Objection.

15 Misstates testimony.

16 THE WITNESS: Again, I don't
17 think that's a doable thing to do
18 because of our field.

19 On the other hand, there have
20 been experts who have looked at my
21 report and made comments. And so I
22 think in terms of that, they have had
23 an understanding of the evidence that
24 I reviewed, of the literature, of the
25 data behind that, and they certainly

1 have responded. So I think there was
2 understanding.

3 QUESTIONS BY MR. TRACEY:

4 Q. Do you know what a Navigation
5 Guide -- do you know what the Navigation
6 Guide is?

7 A. I do not.

8 Q. Do you know what the Cochrane
9 collaboration guide is?

10 A. I do know about Cochrane
11 reports.

12 Q. Okay. Well, yeah, right.
13 But I'm asking a different
14 question. I'm asking whether or not you know
15 about the Cochrane method for evaluating
16 scientific evidence?

17 A. I have formally not performed a
18 Cochrane review.

19 Q. Okay. You have not used the
20 Navigation Guide either, correct?

21 A. I have not used the Navigation
22 Guide.

23 Q. You did not employ an adverse
24 outcome pathway in the -- in your report?

25 A. I did not.

1 Q. Do you know what that is?

2 A. I can guess, but, no, I don't
3 formally know.

4 Q. Okay. Let's -- you have said a
5 number of times here today in your report,
6 the last -- oh, second from the last sentence
7 in the report. Let me just read it because I
8 think it's the punch line of your report.

9 You say in your report -- you
10 can just turn to the last page, page 77,
11 under Conclusion, Doctor.

12 A. Okay.

13 Q. You say, "For the reasons
14 described" -- can we put it on the screen?
15 Does that -- oh, okay. Thanks.

16 "For the reasons described in
17 this report, I conclude that genetics are the
18 predominant cause of ADH" -- "of ASD and
19 ADHD."

20 Right?

21 A. That is what I state.

22 Q. And then, 2, "There is
23 insufficient scientific" -- "scientific
24 evidence to support the conclusion that
25 maternal intake of acetaminophen during

1 pregnancy can cause the development of ASD
2 and ADHD in offspring."

3 Those are your -- that's your
4 one -- two-sentence -- or one-sentence
5 conclusion, right?

6 A. That is correct.

7 Q. What do you mean, Doctor, when
8 you say, "genetics are the predominant cause
9 of ASD and ADHD"?

10 I want to understand that,
11 please.

12 A. So that when -- tries to
13 understand causation or contributors to both
14 of those phenotypes that the overwhelming,
15 underlying contributors are genetics. And as
16 we estimate that, using estimates of
17 heritability, those numbers are extremely
18 high; 80 to 90 percent for both of those
19 conditions.

20 Q. I noticed you use
21 "heritability" in your report quite often,
22 but you never defined it, did you?

23 A. I believe I did.

24 Q. Oh, where? I didn't see it.
25 Please tell me where you define

1 **"heritability."**

2 A. This is easier to do
3 electronically.

4 MS. BROWN: Yeah.

5 QUESTIONS BY MR. TRACEY:

6 Q. And I looked, ma'am. I looked
7 for a definition of heritability. Now, I'm
8 not a techno geek or proficient with it, so I
9 could have missed it.

10 A. On page 20.

11 Q. Okay.

12 A. Under Section 44.

13 Q. **"Heritability is the fraction**
14 **of the variability of the phenotype that is**
15 **due to inherited genetic factors."**

16 **Is that right?**

17 A. That's correct.

18 Q. Are you implying to the Court
19 and everybody that heritability is
20 independent and divorced from environment?

21 A. It is just what I said; that
22 heritability is inherited genetic factors.

23 I will also state that there
24 are other genetic factors that are not
25 inherited, but this is putting a point that

1 the overwhelming majority of the probability
2 of an individual having autism or ADHD, 80 to
3 90 percent of that is due to heritable or
4 inherited genetic factors, in addition to
5 which there are de novo genetic factors,
6 which means that all told, genetics is the
7 overwhelming predominance in terms of
8 contributors.

9 **Q. Doesn't heritability allow a**
10 **comparison of the relative importance of**
11 **genes and environment to the variation of**
12 **traits within and across populations?**

13 A. I'm not sure exactly what you
14 mean by that, but heritability is an estimate
15 of the variance in the phenotype attributable
16 to inherited genetic factors.

17 **Q. Okay. Do you know who Peter**
18 **Visscher is?**

19 A. I do not.

20 **Q. What about Naomi Wray?**

21 A. I do not.

22 **Q. What about William Hill?**

23 A. I do not.

24 (Chung Exhibit 398 marked for
25 identification.)

1 QUESTIONS BY MR. TRACEY:

2 Q. I'm going to hand you
3 exhibit --

4 MR. TRACEY: What's the number,
5 Danny? 319 -- 398, sorry.

6 QUESTIONS BY MR. TRACEY:

7 Q. This is something -- a paper
8 published in Nature Reviews Genetics.
9 You're familiar with this
10 publication, ma'am, are you not?

11 A. I don't recall whether or not
12 I've read this particular paper.

13 Q. No, ma'am. But I was asking
14 you about the publication itself, Nature
15 Reviews Genetics.

16 A. Yes, I know of the journal.

17 Q. It's one of the highest impact
18 journals in the world in the discipline of
19 genetics, isn't it?

20 A. I don't know the current impact
21 factor.

22 Q. Okay. But it's well-respected,
23 is it not?

24 A. It's part of the Nature series
25 of journals.

1 Q. And that's one of the most
2 well-respected journals, Nature, in the
3 world, right?

4 A. This is one of a series in
5 Nature.

6 Q. But isn't Nature one of the
7 most well-respected journals in the world?

8 A. The journal Nature is, but this
9 is not the journal Nature.

10 Q. Yeah. Okay.
11 The very first sentence in the
12 abstract, ma'am, says, "Heritability" --
13 well, let's read the title.

14 "Heritability in the genomics
15 era - concepts and misconceptions."

16 Do you see that?

17 A. I see the title.

18 Q. And the very first sentence in
19 the abstract says, "Heritability allows a
20 comparison of the relative importance of
21 genes and environment to the variation of
22 traits within and across populations."

23 Do you see that?

24 A. Yes, I do see that.

25 Q. Is that a true statement?

1 A. Heritability -- I think the
2 assumption is is that things are not
3 inherited -- or heritable or environment.
4 So, yes, if one thinks about this is
5 heritable and nonheritable, yes, that's true.

6 **Q. That's a true statement, yeah?**

7 A. What I said I believe to be
8 true.

9 **Q. Well, is that statement on the**
10 **screen that's highlighted in yellow true?**

11 A. As I said, if one considers the
12 environment in a very broad way, everything
13 that's nonheritable, yes, that's true.

14 **Q. Okay. And then if you flip**
15 **over -- this entire paper, you will see by**
16 **its title, is about misconceptions with**
17 **respect to heritability. And if you turn**
18 **over to the page where at the top it says,**
19 **"Box 2," it's page 257, you see that box is**
20 **actually titled "Misconceptions regarding**
21 **heritability."**

22 **Do you see that?**

23 A. Yes, I see that.

24 **Q. And then the good doctors list**
25 **various misconceptions that people have about**

1 **heritability.**

2 **Do you see that?**

3 A. I see that they have several
4 things in this box that they've listed.

5 **Q. And this -- yeah.**

6 **And the second thing I want**
7 **to -- the second misconception they identify**
8 **is "high heritability implies genetic**
9 **determination."**

10 **Do you see that?**

11 A. I see that title.

12 **Q. Do you agree with these doctors**
13 **that that is a misconception?**

14 MS. BROWN: And, Doctor, I --

15 THE WITNESS: I haven't --

16 MS. BROWN: -- you testified
17 you haven't read this paper, you don't
18 recall reading it. So if you need a
19 moment to take a look, please do
20 before you answer.

21 THE WITNESS: I'd really, to be
22 fair, need to have time to read the
23 entire -- this review to be able to
24 comment on this.

25

1 QUESTIONS BY MR. TRACEY:

2 Q. Ma'am, you can't answer the
3 question as a geneticist whether it is a
4 misconception of genetics to claim that high
5 heritability implies genetic determination?

6 MS. BROWN: Well, that
7 misstates her testimony. I object.

8 THE WITNESS: I do not agree
9 that high heritability does not mean
10 that there is a high percent of the
11 variation that is due to underlying
12 genetic causes.

13 QUESTIONS BY MR. TRACEY:

14 Q. Okay. Well, let's read on.
15 They go on to say, "A high
16 heritability means that most of the variation
17 that is observed in the present population is
18 caused by variation in genotypes."

19 Do you -- are you with them --
20 do you agree with that so far?

21 A. Again, within this, to have an
22 opinion on this, I'd really need to read
23 through the entire review to be able to
24 understand the entire context with which this
25 was written.

1 Q. Okay. Well, let's read on, see
2 if it becomes any more clear.

3 It says, "It means that in the
4 current population, the phenotype of an
5 individual is a good predictor of the
6 genotype."

7 How about that?

8 MS. BROWN: Well, I just have a
9 running objection to this --

10 MR. TRACEY: You can have it.

11 MS. BROWN: -- to this document
12 that she's not familiar with and is
13 not on --

14 MR. TRACEY: You can have a
15 running objection.

16 MS. BROWN: -- on her reliance
17 list, and she's asked for time to
18 review.

19 THE WITNESS: Again, I mean, if
20 we'd like to give me a couple hours to
21 read this and then to comment on it,
22 we can.

23 I'd prefer not being able -- I
24 want to be accurate in my statements,
25 and I prefer not taking one sentence

1 out of context for the entire review.

2 QUESTIONS BY MR. TRACEY:

3 Q. You can't answer that question
4 without spending a couple of hours with the
5 paper?

6 MS. BROWN: Well, that's not
7 what she said.

8 THE WITNESS: Again --

9 MR. TRACEY: It is what she
10 said.

11 MS. BROWN: No.

12 THE WITNESS: Again, I've given
13 my opinion in terms of genetics and
14 heritability estimates and what that
15 tells us.

16 To be able to take a sentence
17 within this and to accurately
18 interpret what it means, I should read
19 the entirety of this.

20 QUESTIONS BY MR. TRACEY:

21 Q. Well, let's read on and see if
22 we can get some agreement.

23 It says, "However, it does not
24 mean that the phenotype is determined once we
25 know the genotype."

1 **Do you agree with that?**

2 MS. BROWN: Same objections.

3 THE WITNESS: What I agree in
4 general is that it is impossible for
5 us in this day of 2023 to understand
6 the entirety of, quote/unquote, the
7 genotype.

8 Our incomplete understanding of
9 that genotype leads us to imprecision
10 with predicting the genotype. With
11 that statement, I agree.

12 QUESTIONS BY MR. TRACEY:

13 **Q. So they go on to say, "The**
14 **reason it doesn't mean that is because the**
15 **environment can change or be manipulated to**
16 **alter the phenotype."**

17 **That's a true statement, isn't**
18 **it?**

19 MS. BROWN: Same objections.

20 THE WITNESS: Again, I believe
21 that the environment is different for
22 different people and over different
23 periods of time. Being able to
24 correctly model the environmental
25 exposures, there's something that

1 we're currently imprecise at doing.

2 QUESTIONS BY MR. TRACEY:

3 Q. Even if you're imprecise at
4 doing it, there's no question in your field
5 that heritability encompasses both genetics
6 and the environment, correct?

7 MS. BROWN: I object.

8 Misstates testimony.

9 THE WITNESS: Again, I've
10 stated the definition of heritability
11 is the variance -- the percentage of
12 the variance and the phenotype
13 accountable by inherited genetic
14 factors.

15 QUESTIONS BY MR. TRACEY:

16 Q. I know you have, but can you
17 answer my question?

18 A. Can you please restate the
19 question?

20 Q. Yeah.

21 There's no doubt in your field
22 of genetics that heritability includes the
23 environment?

24 A. The phenotype includes the
25 environment.

1 Q. And let's define phenotype.

2 Maybe we ought to do that.

3 What is -- is that the trait or
4 the behavior associated that we're looking
5 at?

6 A. Phenotype is within the context
7 of a particular scientific question.

8 Q. Yeah.

9 So autism spectrum disorder is
10 a phenotype?

11 A. It can be.

12 Q. ADHD is a phenotype?

13 A. It can be.

14 Q. Phenotypes are influenced by
15 the environment?

16 MS. BROWN: Objection to the
17 form.

18 THE WITNESS: Phenotypes, that
19 is a very broad statement.

20 QUESTIONS BY MR. TRACEY:

21 Q. Yes, ma'am.

22 A. And it depends on what the
23 phenotype is, whether that's influenced by
24 the environment or not.

25 Q. Autism is a phenotype that is

1 **absolutely influenced by the environment?**

2 A. I would say that our
3 understanding of autism in the present day is
4 that the overwhelming contributor to autism
5 is genetic, be that either inherited genetic
6 factors or de novo genetic factors, but the
7 overwhelming contribution is genetic.

8 **Q. Okay. Do you want to answer my**
9 **question?**

10 MS. BROWN: Objection.

11 Argumentative.

12 She did her very best to answer
13 the question that you asked.

14 THE WITNESS: At this point in
15 terms of understanding, quote/unquote,
16 the environment -- or I will take that
17 liberally to say nongenetic
18 environment, we do believe that there
19 is a small contribution of those
20 nongenetic factors because that
21 heritability is not one. It is not
22 100 percent.

23 But on the other hand, being
24 that it is 80 to 90 percent, that
25 means that the overwhelming

1 contribution is genetic, and that to
2 be able to implicate something that is
3 environmental must contribute -- must
4 consider those genetic confounds
5 potentially in terms of understanding
6 a nongenetic or environmental
7 contributor.

8 QUESTIONS BY MR. TRACEY:

9 **Q. Does the environment contribute**
10 **to the cause of autism, Dr. Chung?**

11 A. I know this is complicated.

12 **Q. No, no, it's not.**

13 MS. BROWN: Let her --

14 MR. TRACEY: It's not.

15 MS. BROWN: Let her finish,
16 please, sir. She's answering your
17 question.

18 THE WITNESS: So the
19 overwhelming contributor to autism or
20 ADHD is genetic.

21 QUESTIONS BY MR. TRACEY:

22 **Q. Can you answer my question?**

23 A. Given that the overwhelming
24 contributor to both autism and ADHD is
25 genetic, we know that it is not 100 percent

1 genetic, but it is overwhelmingly genetic.

2 And, therefore, to be able to
3 properly assess the evidence for anything
4 that's not genetic must consider genetic
5 contributors to ensure that there are not
6 genetic confounds in whatever nongenetic
7 factors are implicated.

8 **Q. We'll get there. Can you bring**
9 **up Exhibit 319, please?**

10 MS. BROWN: And, Mr. Tracey,
11 after this exhibit, would it be a good
12 time for a break?

13 MR. TRACEY: Sure.

14 (Chung Exhibit 319 marked for
15 identification.)

16 QUESTIONS BY MR. TRACEY:

17 **Q. You know what the National**
18 **Institutes of Environmental Health and**
19 **Sciences is, Dr. Chung?**

20 A. I do.

21 **Q. Have you been on their website**
22 **before?**

23 A. I don't recall.

24 MS. BROWN: Can she have a hard
25 copy of this?

1 MR. TRACEY: Yeah.

2 MS. BROWN: Thank you.

3 QUESTIONS BY MR. TRACEY:

4 Q. I found this on the website.
5 You see that -- the date is three days ago.
6 Do you see that, August 27,
7 2023?

8 A. I see that date.

9 Q. The National Institute of
10 Environmental Health Sciences is a branch of
11 the federal government, right?

12 MS. BROWN: Hang on. We're
13 just getting sorted with our exhibits.

14 THE WITNESS: I'm sorry, can
15 you repeat your question?

16 QUESTIONS BY MR. TRACEY:

17 Q. Do you understand the National
18 Institute of Environmental Health Sciences to
19 be a branch of the federal government?

20 A. It is one of the institutes
21 within NIH.

22 Q. Yeah.
23 Which is supported and funded
24 by the federal government.

25 A. That is correct.

1 Q. Now, what they say, if you go
2 to their website and you have a question
3 about gene and environment interaction, they
4 say, "Few diseases result from a change in a
5 single gene or even multiple genes. Instead,
6 most diseases are complex and stem from an
7 interaction between your genes and your
8 environment."

9 Do you agree with the NI --
10 National Institutes of Environmental Health
11 Sciences?

12 A. That's a very broad statement,
13 and I agree that when one thinks about many
14 conditions -- and, again, there are many,
15 many, hundreds, thousands, of different
16 conditions -- that is a very broad statement.

17 Q. They go on -- it is, ma'am.
18 Trust me, we're going to get specific soon.

19 It says, "Factors in your
20 environment can range from chemicals in the
21 air or water pollution, mold, pesticides,
22 diet choices or grooming products."

23 Do you agree with that?

24 A. I believe that it's a statement
25 that they've made. I think one needs to get

1 to specificity to understand particular
2 conditions.

3 Q. Okay. They go on to say,
4 "Subtle differences in one person's genes can
5 cause them to respond differently to the same
6 environmental exposure as another person."

7 Do you agree with that?

8 A. Again, realizing that this is a
9 very broad statement about many different
10 conditions and many different exposures,
11 there are some differences between
12 individuals.

13 Q. That's called, what, genetic
14 susceptibility?

15 A. Genetic susceptibility is a
16 portion of that statement.

17 Q. Okay. They go on to say, "As a
18 result, some people may develop a disease
19 after being exposed to something in the
20 environment while others may not."

21 And that's a true statement,
22 right?

23 A. Again, this is a very broad
24 statement, and it depends in terms of what
25 condition and what environmental exposure.

1 Q. Okay. Scroll down there,
2 please, Mike.

3 Now, the next section I want to
4 point to is, "What is the NIEHS doing?"

5 It says, "NIEHS studies a wide
6 range of diseases and disorders with genetic
7 and environmental component."

8 Did you know that they did
9 that?

10 A. Yes, I was aware.

11 Q. It says, "In addition, new
12 technologies and computational approaches are
13 under development to tease out the gene and
14 environment interactions that underpin
15 disease."

16 Did you know that was going on?

17 A. Yes, I was aware.

18 Q. And then the very first disease
19 they flag for us is autism.

20 Do you see that?

21 A. They do have a bullet under
22 autism.

23 Q. Remember I said we were going
24 to get specific?

25 A. Yes, I recall.

1 Q. They say, "High levels of air
2 pollution increase the risk for autism in
3 children with a genetic variant called MET,
4 which is involved in brain development."

5 Do you know whether that's
6 true?

7 A. I don't know about that
8 particular gene.

9 Q. They say, "This genetic variant
10 did not increase the risk for the 75 percent
11 of the population exposed to lower levels of
12 air pollution, suggesting that autism may be
13 caused by an interaction of genetic and
14 environmental factors."

15 Did you know that?

16 A. I believe they're referencing
17 one paper specifically about one genetic
18 variance and interaction with one particular
19 exposure of air pollution.

20 Q. Okay. Do you disagree with
21 them? Are they just wrong?

22 A. Can you be precise in what
23 you're asking if I disagree about?

24 Q. Well, you're seeming to imply
25 that maybe they're incorrect.

1 **Am I wrong in getting that**
2 **impression?**

3 MS. BROWN: Objection to the
4 form of the question.

5 QUESTIONS BY MR. TRACEY:

6 **Q. If I'm wrong, just tell me I'm**
7 **wrong.**

8 A. I just like to be precise in
9 what we're -- what statement specifically,
10 we -- we've listed several statements.

11 **Q. Well, the statement that we**
12 **just read into the record that "This genetic**
13 **variant did not increase the risk for**
14 **75 percent of the population exposed to lower**
15 **levels of air pollution," suggesting that**
16 **autism may be caused by an interaction of**
17 **genetic and environmental factors, that**
18 **statement.**

19 A. I'll timestamp when this
20 particular paper was published, which was in
21 2014, which is already almost a decade ago,
22 which in terms of genetic history is ancient.

23 And so within this, although
24 this may have at the time been something that
25 was believed, in many cases we're going back

1 and revisiting with much more complicated
2 methods, models, being able to consider
3 multiple contributors.

4 I don't know whether or not
5 these investigators have readdressed this
6 same question using currently available
7 technologies and methods.

8 **Q. The date that I pulled this off**
9 **the Internet was three days ago, though, not**
10 **nine years ago, right?**

11 A. That's correct, but I'm
12 referencing the primary literature that they
13 refer to, and I'm commenting on the primary
14 literature. I'm not commenting on the
15 science communicator that wrote their
16 website.

17 **Q. Well, you're not really**
18 **commenting on the primary literature. What**
19 **you're doing is saying that it's nine years**
20 **old.**

21 **Are you testifying that that**
22 **statement is false based on new and improved**
23 **technologies?**

24 MS. BROWN: I object. That's
25 argumentative.

1 THE WITNESS: Again, what I'm
2 stating is that in terms of the
3 question, I'm not aware if these
4 investigators have done the most
5 up-to-date analysis that's possible to
6 do that considers the totality of the
7 evidence to understand the
8 contributors.

9 QUESTIONS BY MR. TRACEY:

10 Q. Okay. All right. You're
11 ignorant of whether they've done further
12 research?

13 MS. BROWN: Objection to the
14 form.

15 THE WITNESS: I'm ignorant. I
16 don't know of all research done in all
17 aspects.

18 QUESTIONS BY MR. TRACEY:

19 Q. Okay. That's not my question.
20 My question is, are you
21 claiming that what's on this page is
22 scientifically false?

23 MS. BROWN: Objection to the
24 form of the question.

25 THE WITNESS: Again, to be

1 precise, can we talk about which
2 sentence specifically we're talking
3 about?

4 QUESTIONS BY MR. TRACEY:

5 **Q. The same sentence I read in the**
6 **last time you asked me two minutes ago --**

7 A. And I'm sorry --

8 **Q. -- this genetic variant.**

9 A. Again, I believe this website
10 is referencing this paper, this primary
11 literature, from 2014, and I believe this
12 website is accurately reflecting what is
13 written in this paper in 2014.

14 I am saying I have not
15 specifically gone back, and I don't know if
16 these authors have gone back, to check the
17 validity of this statement or this
18 information given current methods that we
19 have today.

20 **Q. I understand you're ignorant,**
21 **Doctor, of what's been done since then, but I**
22 **want an answer to a different question.**

23 A. So what I see --

24 **Q. I want to know -- let me -- let**
25 **me ask it again.**

1 I want to know whether Wendy
2 Chung today, August the 30th, 2023, is
3 testifying that that sentence that we've read
4 now twice into the record is false?

5 MS. BROWN: I object. She's
6 answered this question at least three
7 times now.

8 THE WITNESS: I'm not saying
9 whether this sentence is either true
10 or false.

11 QUESTIONS BY MR. TRACEY:

12 Q. Okay. I'm going to hand you
13 Exhibit 312 --

14 MS. BROWN: Can we do a break
15 before that?

16 MR. TRACEY: Oh, yeah, sorry.
17 Yeah. Yeah.

18 VIDEOGRAPHER: The time is
19 10:00 a.m., and we're off the record.
20 (Off the record at 10:00 a.m.)

21 VIDEOGRAPHER: The time is
22 10:11 a.m., and we're on the record.

23 (Chung Exhibit 312 marked for
24 identification.)
25

1 QUESTIONS BY MR. TRACEY:

2 Q. Dr. Chung, on the break, I
3 walked down -- the next exhibit, it's -- what
4 is the number? Is it 312?

5 Sorry, 312, it's an article
6 called "Neurobehavioral Effects of
7 Developmental Toxicity," published in Lancet
8 Neurology, it says 2014 up there, but I think
9 it was ultimately published in 2015.

10 Are you familiar with the
11 journal Lancet Neurology?

12 A. I'm familiar with Lancet
13 Neurology.

14 Q. Do you -- do you understand
15 that to be one of the premium journals,
16 scientific journals, in the world?

17 A. I don't know the impact factor.
18 Lancet certainly -- Lancet properly is a
19 leading journal.

20 Q. Okay. Do you know who either
21 of the authors are, Philippe Grandjean or
22 Philip Landrigan?

23 A. I personally don't know either
24 of them.

25 Q. Well, have you heard of either

1 **one of the them?**

2 A. Off the top of my head, no.

3 **Q. Dr. Grandjean is, it says, with**
4 **the Department of Environmental Medicine,**
5 **University of Southern Denmark and also an**
6 **appointment at the Department of**
7 **Environmental Health at the Harvard School of**
8 **Public Health here in Boston.**

9 **Do you see that?**

10 A. I see that with his
11 affiliations.

12 **Q. And then Dr. Landrigan, at**
13 **least at the time, was at the Icahn School of**
14 **Medicine in Mount Sinai, New York, correct?**

15 A. That's what's listed, yes.

16 **Q. Those are two institutions of**
17 **impeccable scientific quality and integrity,**
18 **right?**

19 A. I think you have to not overly
20 generalize for institutions and talk about
21 individual investigators, but I don't know
22 either of them.

23 **Q. Okay. What is Harvard's**
24 **reputation, the Department of Environmental**
25 **Health at Harvard? Is it -- does it enjoy a**

1 **fine reputation worldwide?**

2 A. I assume it does have a fine
3 reputation. Again, more importantly are the
4 individual investigators.

5 Q. Okay. Let's see what these two
6 individual investigators that published in
7 Lancet have to say about the subject of
8 neurobehavioral effects of developmental
9 toxicity, shall we?

10 If you -- if you scroll down,
11 the last sentence in the abstract, it says,
12 "To control the pandemic of developmental
13 neurotoxicity, we propose a global prevention
14 strategy. Untested chemicals should not be
15 presumed to be safe to brain development, and
16 chemicals in existing use and all new
17 chemicals must therefore be tested for
18 developmental neurotoxicity."

19 Now, I have a couple questions
20 about that.

21 Do you see them describe a
22 pandemic of developmental neurotoxicity?

23 MS. BROWN: Well, I -- same
24 objection as before. She hasn't
25 reviewed this article, and so she

1 needs time to look at it to answer
2 your question.

3 QUESTIONS BY MR. TRACEY:

4 Q. Ma'am, do you agree that --
5 with these authors, that there is a pandemic
6 of developmental neurotoxicity that needs to
7 be controlled?

8 MS. BROWN: Same objection.

9 THE WITNESS: Again, I haven't
10 read this particular paper. As we're
11 thinking about the world around us, I
12 would not describe this as a pandemic.

13 QUESTIONS BY MR. TRACEY:

14 Q. Okay. Do you agree that
15 untested chemicals should not be presumed to
16 be safe to brain development?

17 A. There are literally hundreds of
18 thousands of chemicals, so that's a very,
19 very broad statement.

20 Q. It is, ma'am.
21 Do you agree with it?

22 MS. BROWN: Object to the form.

23 THE WITNESS: Again, I think it
24 would be impossible to test as
25 chemicals as exist -- the number of

1 chemicals that exist on the face of
2 the earth to be able to test for
3 developmental neurotoxicity.

4 QUESTIONS BY MR. TRACEY:

5 **Q. It would be impossible?**

6 A. I don't believe that people
7 would put the resources necessary to be able
8 to do this and to be able to do it in an
9 ethical way.

10 **Q. Okay. Have you yourself**
11 **studied or published on acetaminophen?**

12 A. I don't believe so.

13 **Q. You don't have any papers on**
14 **acetaminophen and neurotoxicity?**

15 A. I believe I may have a paper of
16 acetaminophen with breast cancer, if I have a
17 paper.

18 **Q. Okay. But I was asking about**
19 **neurotoxicity.**

20 A. To the best of my recollection,
21 I do not.

22 **Q. Okay. Do you know, or did you**
23 **run across in your research for this case,**
24 **the developmental neurotoxicity tests that**
25 **were done on acetaminophen before it was --**

1 **started getting sold to the public?**

2 MS. BROWN: Objection to the
3 form.

4 THE WITNESS: I did not review
5 such literature.

6 QUESTIONS BY MR. TRACEY:

7 **Q. Did you ask anybody if such**
8 **literature existed before you rendered your**
9 **opinions in this case?**

10 A. I did not ask specifically for
11 that literature.

12 **Q. Do you know if it even exists?**

13 A. I did not do a comprehensive
14 literature review for that specific question.
15 I did a comprehensive literature review, as
16 we've talked about before, thinking about
17 prenatal acetaminophen, autism and ADHD.

18 **Q. But my question -- I understood**
19 **that.**

20 But my question is, do you even
21 **know if the literature on developmental**
22 **neurotoxicity and acetaminophen has been**
23 **done?**

24 MS. BROWN: Objection. Asked
25 and answered.

1 THE WITNESS: I did not do a
2 search specifically with the terms
3 that you just mentioned, so I can't
4 comment on what literature is
5 available using those terms
6 specifically.

7 QUESTIONS BY MR. TRACEY:

8 Q. So you don't have any idea, as
9 we sit here today, whether it exists or it
10 doesn't?

11 MS. BROWN: Asked and answered.
12 Objection.

13 THE WITNESS: Again, I was
14 thinking about the data that we have
15 to support, from an epidemiological
16 and a genetic point of view, the role
17 of acetaminophen.

18 QUESTIONS BY MR. TRACEY:

19 Q. Can you answer my question?

20 A. Again, because I did not do the
21 literature review that you mentioned, I can't
22 comment on what the data are or are not.

23 Q. Okay. Do you know whether
24 Tylenol was never tested for developmental
25 neurotoxicity before it began being marketed

1 **in the United States?**

2 MS. BROWN: Object to the form.

3 Lacks foundation.

4 QUESTIONS BY MR. TRACEY:

5 **Q. Has anybody ever told you that?**

6 MS. BROWN: I object. That

7 lacks foundation.

8 THE WITNESS: Again, for

9 acetaminophen, I'm not aware of

10 published or unpublished data using

11 the terms that you've just used of

12 neurotoxicity.

13 QUESTIONS BY MR. TRACEY:

14 **Q. Do you know whether or not**

15 **Tylenol, acetaminophen, is hepatotoxic in**

16 **humans?**

17 A. Certainly I do know of the

18 hepatotoxicity of acetaminophen.

19 **Q. Do you know how long**

20 **acetaminophen was being sold worldwide before**

21 **it was discovered that acetaminophen was**

22 **hepatotoxic?**

23 A. I do not know the answer to

24 that specific question.

25 MS. BROWN: Object, I --

1 belated objection to that question.

2 QUESTIONS BY MR. TRACEY:

3 Q. You don't know whether it'd
4 been -- it had been being sold for months,
5 years or decades before that was discovered?

6 A. I don't know.

7 Q. Okay. Do you know how many
8 years it had been marketed worldwide to the
9 public as an over-the-counter medicine before
10 a warning was put on it regarding
11 hepatotoxicity?

12 MS. BROWN: Objection to the
13 form. Lacks foundation.

14 THE WITNESS: I don't know.

15 QUESTIONS BY MR. TRACEY:

16 Q. Do you know whether or not
17 Tylenol, acetaminophen, is the leading cause
18 of accidental liver overdose deaths in the
19 world?

20 MS. BROWN: Objection to the
21 form. Lacks foundation.

22 THE WITNESS: I don't know. I
23 haven't specifically investigated that
24 question.

25

1 QUESTIONS BY MR. TRACEY:

2 Q. All right. Well, let's read on
3 Landrigan and Grandjean.

4 At the bottom of the
5 introduction, the sentence that says, "The
6 root causes of the present global pandemic of
7 neurodevelopmental disorders" -- oh, sorry.
8 I skipped something. Sorry.

9 First sentence under
10 Introduction. It says, "Disorders of
11 neurobehavioral development affect 10 to
12 15 percent of all births, and prevalence
13 rates of autism spectrum disorder and
14 attention-deficit/hyperactivity disorder seem
15 to be increasing worldwide."

16 Do you agree with that?

17 MS. BROWN: Objection to the
18 form of the question.

19 THE WITNESS: I think it
20 depends on where you are in the world
21 and where you are over time, but at
22 the time that this paper was published
23 in 2015, I believe those estimates
24 were approximately correct.
25

1 QUESTIONS BY MR. TRACEY:

2 Q. All right. Next paragraph.

3 "The root causes of the present global
4 pandemic of neurodevelopmental disorders are
5 only partly understood."

6 You certainly agree with that?

7 A. That is true.

8 Q. It says, "Although genetic
9 factors have a role, they cannot explain," on
10 the next page, "increases in reported
11 prevalence, and none of the genes discovered
12 so far seem to be responsible for more than a
13 small proportion of cases."

14 Do you see that?

15 MS. BROWN: So same objection
16 to reading sentences out of this
17 article she hasn't had time to review.

18 QUESTIONS BY MR. TRACEY:

19 Q. Do you agree with that?

20 A. I don't agree with that.

21 Q. Okay.

22 A. I do see the sentence that
23 you've highlighted here. I'll again say that
24 this is timestamped, published, 2015,
25 probably written in 2014, and our state of

1 knowledge of genetics over that almost decade
2 has been remarkably different.

3 And specifically when it comes
4 to de novo genetic factors, those have
5 changed over time as reproductive patterns
6 have changed over time.

7 Q. I assure you, Dr. Chung, we're
8 going to come forward in time up till and
9 including today.

10 All right?

11 MS. BROWN: Let's just ask
12 questions.

13 QUESTIONS BY MR. TRACEY:

14 Q. Is that okay with you, Doctor?

15 A. I'm ready for the next
16 question.

17 Q. All right. These doctors say,
18 "Overall, genetic factors seem to account for
19 no more than perhaps 30 to 40 percent of all
20 cases of neurodevelopmental disorders. Thus,
21 nongenetic, environmental exposures are
22 involved in causation, in some cases probably
23 by interacting with genetically inherited
24 predispositions."

25 Do you see that?

1 MS. BROWN: Same objections.

2 THE WITNESS: I see that,
3 although I haven't read this
4 particular paper.

5 I'll point out that what they
6 reference in terms of this very broad
7 group of neurodevelopmental disorders
8 is not specific, and the statements
9 that you've just read are not specific
10 either to autism or ADHD in what
11 you've read.

12 QUESTIONS BY MR. TRACEY:

13 Q. You're absolutely right. I
14 just read what I read.

15 Right?

16 A. Correct, you read what they
17 wrote.

18 Q. That sounds strikingly similar,
19 that last sentence, though, to what the
20 National Institutes of Environmental Health
21 Sciences has on their website as of three
22 days ago, doesn't it?

23 MS. BROWN: Objection to the
24 form.

25 THE WITNESS: Again, what the

1 National Institutes of Environmental
2 Health Sciences has written is quite
3 broad in their statements, and we need
4 to get down to the specificity of the
5 issues related to autism and ADHD.

6 QUESTIONS BY MR. TRACEY:

7 Q. Okay. And the next paragraph,
8 the second sentence, it says, "The developing
9 human brain is uniquely vulnerable" --

10 A. I'm sorry, where are we?

11 Q. Sorry. The second sentence of
12 the next paragraph. We're going to highlight
13 it for you. It may be easier to follow along
14 on the screen.

15 A. Okay.

16 Q. "The developing human brain is
17 uniquely vulnerable to toxic chemical
18 exposures, and major windows of developmental
19 vulnerability occur in utero and during
20 infancy and childhood."

21 Do you agree with that?

22 A. I agree with the statement
23 that's written here that, again, we're
24 talking about in a very general way --

25 Q. Yes, ma'am.

1 A. -- many conditions.

2 **Q. They go on to say that, "During**
3 **these sensitive life stages, chemicals can**
4 **cause permanent brain injury at low levels of**
5 **exposure that would have little or no adverse**
6 **effect in an adult."**

7 **Do you agree with that?**

8 A. Again, these are very, very
9 general statements in terms of hundreds of
10 thousands of chemicals that could be at play
11 without any level of specificity.

12 **Q. Okay. But generally speaking,**
13 **you agree with that statement?**

14 A. I believe that there are
15 different life stages and different things
16 happen at different life stages; fetal
17 development being different than newborn
18 development being different than adults. But
19 that's a very -- again, the statements that
20 you've read so far are extremely broad.

21 **Q. And if you skip down two**
22 **paragraphs, the one starting "We noted," it**
23 **says, "We noted that recognition of the risk**
24 **of industrial chemicals to brain development**
25 **has historically needed decades of research**

1 and scrutiny, as shown in the cases of lead
2 and methylmercury."

3 Are you familiar, Dr. Chung,
4 with the history of methylmercury and lead in
5 this country?

6 A. I'm more --

7 MS. BROWN: Objection. Vague.

8 THE WITNESS: I'm more familiar
9 with the lead and what we do in terms
10 of monitoring lead levels in children.

11 QUESTIONS BY MR. TRACEY:

12 Q. Okay. They go on to say, "In
13 most cases, discovery began with the clinical
14 diagnosis of poisoning in workers and
15 episodes of high-dose exposure. More
16 sophisticated epidemiological studies
17 typically began only much later."

18 Let me ask you this. Have you
19 ever asked anybody when Tylenol began being
20 sold in the United States? What year?

21 A. I have not.

22 Q. Do you have even a vague idea
23 of how long it's been on the market?

24 A. I have not, but at least for
25 decades.

1 **Q. Okay. Do you know why or if**
2 **the sales of Tylenol increased in the 1980s;**
3 **and if so, for what reason?**

4 MS. BROWN: Objection. Lacks
5 foundation.

6 THE WITNESS: I do not. I have
7 not studied sales of acetaminophen.

8 QUESTIONS BY MR. TRACEY:

9 **Q. You're younger than me, but**
10 **when I was a kid, we used to get aspirin.**

11 Do you remember that or have
12 any knowledge of that?

13 A. I'm familiar --

14 MS. BROWN: Wait. Does she
15 have knowledge of what happened when
16 you were a kid? That's the question?

17 QUESTIONS BY MR. TRACEY:

18 **Q. Yeah. When I was a kid,**
19 **aspirin was given to children.**

20 Do you have any knowledge of
21 that?

22 You and I were kids at
23 different times.

24 MS. BROWN: I object.

25 THE WITNESS: I am familiar

1 with the former use of aspirin in
2 children.

3 QUESTIONS BY MR. TRACEY:

4 Q. And the reason we don't give it
5 any more is because it was associated with
6 Reye's syndrome in children, right?

7 MS. BROWN: Objection to the
8 form. This all lacks foundation.

9 THE WITNESS: Yes.
10 Prescription pattern -- or not
11 prescription. Usage patterns have
12 changed over time to try and minimize
13 the impact of Reye's syndrome in
14 children.

15 QUESTIONS BY MR. TRACEY:

16 Q. And how long was aspirin sold,
17 if you know, before we figured out it was
18 causing Reye's syndrome in children?

19 A. I do not know. I haven't
20 investigated that point in particular.

21 Q. If I told you it was sold for
22 over a hundred years, would that surprise
23 you?

24 MS. BROWN: Objection to the
25 form. Lacks foundation.

1 THE WITNESS: I don't know the
2 answer.

3 QUESTIONS BY MR. TRACEY:

4 Q. Aspirin, of course, is -- has
5 been always, for the most part, an
6 over-the-counter product, right?

7 A. Again, I don't have a comment.
8 I don't know.

9 Q. Okay. They go on to say,
10 "Thus, recognition of widespread subclinical
11 toxicity often did not occur until decades
12 after the initial evidence of neurotoxicity."

13 They say, "A recurring theme
14 was that the early warnings of subclinical
15 neurotoxicity were often ignored or even
16 dismissed."

17 Are you familiar, ma'am, with
18 the history of some of the most notorious
19 toxins in our country?

20 MS. BROWN: Objection to the
21 form.

22 THE WITNESS: That's a pretty
23 general statement.

24 QUESTIONS BY MR. TRACEY:

25 Q. Let me give you some specifics.

1 **Are you familiar with the**
2 **history of asbestos poisoning in this**
3 **country?**

4 MS. BROWN: Objection to the
5 form.

6 THE WITNESS: In a very broad
7 sense.

8 QUESTIONS BY MR. TRACEY:

9 **Q. Okay. Do you know who Irving**
10 **Selikoff was?**

11 A. I do not.

12 **Q. Do you know who Richard Doll**
13 **was?**

14 A. I do not.

15 **Q. Are you aware of the research**
16 **linking smoking to lung cancer performed by**
17 **Austin Bradford Hill and Richard Doll in**
18 **1950?**

19 A. I know generally of the
20 studies.

21 **Q. And you know that those were**
22 **observational studies, correct?**

23 A. I have not gone back and
24 reviewed that literature in detail.

25 **Q. Do you know what the regulators**

1 **and the public -- what industry and the**
2 **regulators did to Richard Doll when he told**
3 **the industry and the regulators that smoking**
4 **caused lung cancer?**

5 MS. BROWN: How does this have
6 anything to do with the issues we're
7 here to discuss?

8 I object. This is completely
9 irrelevant. It completely lacks
10 foundation. And it's a huge waste of
11 time.

12 THE WITNESS: I'm not familiar
13 with all of the details. It was
14 something that by the time I trained
15 in medicine and science was already a
16 well-established association.

17 QUESTIONS BY MR. TRACEY:

18 **Q. You're not aware that he was --**
19 **Richard Doll was viciously attacked by both**
20 **regulators and industry when his study was**
21 **published?**

22 MS. BROWN: This completely
23 lacks foundation. I object.

24 THE WITNESS: Again, I -- this
25 is not an area of focus for my

1 research, so I'm not familiar with the
2 details.

3 QUESTIONS BY MR. TRACEY:

4 Q. Do you know how long after his
5 seminal study was published in 1950 that it
6 took to put a warning on cigarettes in the
7 United States?

8 MS. BROWN: Same objections.

9 This is such a waste of time.

10 Irrelevant. Lacks foundation.

11 THE WITNESS: Again, I have not
12 reviewed the history of this
13 particular case study.

14 QUESTIONS BY MR. TRACEY:

15 Q. Do you know what happened to
16 Irving Selikoff when he discovered that
17 asbestos causes cancer?

18 MS. BROWN: Objection.

19 THE WITNESS: I do not.

20 QUESTIONS BY MR. TRACEY:

21 Q. Do you know whether he was
22 vilified, both by regulators and industry, in
23 the press?

24 MS. BROWN: Objection. False
25 and lacks foundation.

1 THE WITNESS: Again, I'm not
2 familiar with the details of this
3 particular case.

4 QUESTIONS BY MR. TRACEY:

5 Q. Does anyone doubt today, ma'am,
6 that smoking causes lung cancer?

7 A. I believe it's a commonly
8 accepted medical association and has resulted
9 in trying, from a public health point of
10 view, to minimize exposures.

11 Q. Does anybody doubt in the
12 scientific community that asbestos causes
13 cancer?

14 MS. BROWN: Objection to the
15 form.

16 THE WITNESS: That's, again, a
17 very broad statement. There are
18 associations between asbestos and
19 particular types of cancer.

20 QUESTIONS BY MR. TRACEY:

21 Q. Let's say mesothelioma.

22 A. I believe that association is
23 well-established.

24 Q. Let's read on.

25 "The good doctors say that

1 **David P. Rall, former director of the US**
2 **National Institute of Environmental Health**
3 **Sciences" --**

4 **That's the outfit we were just**
5 **looking at, right?**

6 A. That is the NIEHS, correct.

7 Q. "David Rall once noted that 'if
8 thalidomide had caused a ten-point loss of
9 intelligence quotient instead of obvious
10 birth defects of the limbs, it would probably
11 still be on the market.'"

12 **Do you see that?**

13 A. I see that sentence.

14 Q. **Do you believe that thalidomide**
15 **causes neurodevelopmental disorders?**

16 MS. BROWN: Objection to the
17 form.

18 THE WITNESS: Thalidomide is
19 not something taken during pregnancy
20 anymore, so I -- it's very difficult
21 to know exactly what the associations
22 are.

23 QUESTIONS BY MR. TRACEY:

24 Q. Well, there's plenty -- there's
25 **studies on thalidomide and birth defects,**

1 **ma'am, are there not, in the historical work?**

2 MS. BROWN: Objection to the
3 form.

4 THE WITNESS: There
5 are associate -- well-published
6 studies of thalidomide and structural
7 congenital anomalies.

8 QUESTIONS BY MR. TRACEY:

9 **Q. And not just phocomelia, which**
10 **is the missing limbs, right?**

11 A. Again, thalidomide was not the
12 focus of my investigation, but thalidomide is
13 not something commonly used by pregnant
14 women.

15 **Q. And the reason it's not**
16 **commonly used by pregnant women is because it**
17 **causes neurodevelopmental disorders and**
18 **structural birth defects?**

19 A. My understanding of the history
20 in reviewing this was because of the
21 association with the limb anomalies.

22 **Q. Okay. Do you know whether or**
23 **not the data supports the fact that**
24 **thalidomide causes autism or**
25 **neurodevelopmental disorders?**

1 MS. BROWN: Objection to the
2 form.

3 THE WITNESS: I did not
4 specifically do a comprehensive review
5 for thalidomide and neurodevelopmental
6 disorders.

7 QUESTIONS BY MR. TRACEY:

8 Q. I'm going to get you to flip
9 back to something just to identify it, and
10 we're going to come back to it.

11 But I just want to flag for
12 your attention on page 19, it's Figure 1,
13 Grandjean and Landrigan have put together a
14 diagram.

15 Do you see that, ma'am --

16 A. I do.

17 Q. -- called "The effect of
18 neurotoxicants during early brain
19 development"?

20 A. I see the figure.

21 Q. And they've got arrows. It
22 starts out "from early life exposures to
23 neurotoxic chemicals," on the left. Got a
24 big arrow pointing to "development/
25 programming."

1 Do you see that, ma'am?

2 A. I see the figure.

3 Q. And then an arrow going down to
4 functional maturation and then ending up at
5 neurological disease and degenerative
6 changes.

7 Do you see that?

8 A. I see the figure, yes.

9 Q. Does that make sense to you,
10 this diagram? Is it understandable to you
11 scientifically?

12 MS. BROWN: Objection to the
13 form. Vague.

14 THE WITNESS: I understand what
15 they're showing in the figure.

16 What they have not shown are
17 all of the other factors contributing
18 to this process, most importantly
19 genetics and genomics.

20 And that is the overwhelming
21 driver that also has a role through
22 what they have talked about in terms
23 of developmental programming,
24 functional maturation and ultimately
25 neurological disease and degenerative

1 changes.

2 And again, as they have
3 referred to this figure, this is a
4 general figure that is not specific in
5 my understanding, without reading the
6 manuscripts, of autism or ADHD but to
7 neurodevelopmental disorders
8 generically.

9 QUESTIONS BY MR. TRACEY:

10 Q. Fair enough.

11 Who's Heather Volk?

12 A. I don't know.

13 Q. You don't know who Heather Volk
14 is at Johns Hopkins University, a researcher?

15 A. I don't.

16 (Chung Exhibit 318 marked for
17 identification.)

18 QUESTIONS BY MR. TRACEY:

19 Q. Interesting. Pull up -- let me
20 have Exhibit 318.

21 I think you're contributing to
22 one of her investigations. That's why I'm
23 surprised. Okay. Here's Exhibit 318. Two
24 copies.

25 This Exhibit 318, as you can

1 **see, Dr. Chung, is called "Considering toxic**
2 **chemicals in the etiology of autism,"**
3 **published in Pediatric Perspectives, not in**
4 **2014 or '15, but in January of 2022.**

5 **Do you see that, ma'am?**

6 A. I see the year of publication,
7 yes.

8 Q. Now, Heather Volk, let's just
9 identify her over on the right. It says she
10 is the Wendy Klag Center -- or with the
11 Wendy Klag Center for Autism and
12 Developmental Disabilities Research,
13 Department of Mental Health, Bloomberg School
14 of Public Health, at Johns Hopkins University
15 in Baltimore, Maryland.

16 **Do you see that?**

17 A. I see the affiliation.

18 Q. You've heard of Johns Hopkins?

19 A. I have.

20 Q. Is that an excellent academic
21 institution?

22 A. The institution is a good
23 institution, and as we've reviewed before, we
24 should also consider the individual
25 investigators, not just the institution.

1 Q. No question. We'll get to that
2 soon enough.

3 I'll also highlight Dr. Beate
4 Ritz, MD, Ph.D., who's the chair of
5 epidemiology in environmental health and
6 neurological at the Fielding School of Public
7 Health at UCLA.

8 Are you familiar with Dr. Ritz
9 or her publications?

10 A. She's not someone I'm
11 intimately familiar with her entire records
12 of publications, no.

13 Q. In your review of the
14 epidemiology literature in this case, do you
15 remember running across her name as being a
16 coauthor on some of the seminal studies on
17 acetaminophen and neurodevelopmental
18 disorders?

19 A. She may have been a coauthor.
20 I don't recall that she was either first or
21 senior author.

22 Q. Okay. They say -- these
23 authors -- this -- and then there's many
24 more, of course, right?

25 A. There are certain many other

1 authors on this paper.

2 Q. Yeah.

3 Looks like they're from all
4 over the country, doesn't it? Pennsylvania,
5 another one from California, right?

6 A. Certainly as with many papers,
7 they have affiliations with many
8 institutions.

9 Q. And what these authors say --
10 the very first sentence, it says, "Diagnoses
11 of autism spectrum disorder, also referred
12 here as" -- oh, yeah -- "ASD also referred to
13 here as autism, a condition that arises
14 during early brain formation, have increased
15 to now 1 in 54 children in the United
16 States."

17 And I think she's citing the
18 CDC for that.

19 Does that sound like an
20 accurate statement to you?

21 A. I believe those were based on
22 CDC prevalence numbers at the time of
23 publication, yes.

24 Q. Now, the next paragraph says,
25 "Scientists long-recognize that genetic

1 **factors contribute to autism etiology as**
2 **indicated in family, twin and genetic**
3 **studies."**

4 **You agree with that, right?**

5 A. Yes, I believe that genetics
6 contribute to autism.

7 They go on to say, "Yet twin
8 studies, from which heritability estimates
9 are primarily derived, may inflate the role
10 of genetics as both gene-only and
11 gene-by-shared environment influences are
12 summarized as genetic."

13 Do you see what the good doctor
14 said there?

15 MS. BROWN: Objection to the
16 form.

17 THE WITNESS: I see what the
18 doctor said there.

19 I'll also restate that
20 heritability estimates don't include
21 all genetic contributors, and de novo
22 genetic variants are not included
23 within that.

24 QUESTIONS BY MR. TRACEY:

25 **Q. I assure you we're going to get**

1 **to de novo, Doctor.**

2 **But do you agree with that**
3 **statement as written?**

4 A. As I've stated before,
5 heritability estimates are a good estimate of
6 genetic contributions to the conditions. The
7 genetics are complicated, but they are a good
8 overall estimate of the aggregate of
9 inherited genetic factors.

10 **Q. But you didn't mention**
11 **environment in your answer, Doctor, like she**
12 **did in her sentence.**

13 A. I will say broadly, again, that
14 there are genetic and I'll call environment
15 nongenetic factors, and because the
16 heritability is not 100 percent, there is
17 something in terms of nongenetic factors, at
18 least not -- not -- at least not heritable
19 genetic factors.

20 **Q. Okay. I'm a little confused.**
21 **Do you agree with that sentence**
22 **as written?**

23 A. I do not agree with this
24 sentence as written.

25 I do agree with the first

1 sentence that you stated, which is that it is
2 long-recognized that genetic factors
3 contribute to autism etiology.

4 **Q. Right.**

5 **But I'm on the next sentence,**
6 **"Yet twin studies from which heritability**
7 **estimates are primarily derived, may inflate**
8 **the role of genetics as both gene-only and**
9 **genetic-by-shared environment influences are**
10 **summarized as genetic."**

11 **That's the sentence I want to**
12 **know if you agree with.**

13 **A. I'll underscore that they use**
14 **the term "may inflate." And within that, I**
15 **would say that I don't believe they do**
16 **inflate, but they are equivocating, I would**
17 **say, in their statement and in that sentence.**

18 **Q. So agree? Disagree?**

19 **MS. BROWN: Asked and answered.**

20 **THE WITNESS: Again, I would**
21 **say that heritability studies -- or**
22 **twin studies, rather, are a good**
23 **estimate of heritability.**

24 **QUESTIONS BY MR. TRACEY:**

25 **Q. And environment?**

1 A. Twin studies are a good
2 estimate of heritability.

3 **Q. And environment?**

4 A. Again, I will state what I
5 stated, my understanding and my opinion,
6 which is that twin studies are a good
7 estimate of heritability.

8 **Q. Well, the twins are in the same**
9 **environment, aren't they?**

10 MS. BROWN: Objection to the
11 form. Lacks foundation.

12 THE WITNESS: They may or may
13 not be.

14 QUESTIONS BY MR. TRACEY:

15 **Q. Well, the twins -- in utero**
16 **twins born vaginally are in the same in utero**
17 **environment?**

18 A. Again, you have just specified
19 something different, which is in utero
20 environment, but there's also a postnatal
21 environment.

22 **Q. Okay. All right. Let's move**
23 **on.**

24 **Dr. Volk and her coauthors say,**
25 **"This pervasive problem of identifying**

1 genetic contributions and assuming their
2 effects cannot result from -- from genes
3 acting in concert with environmental agents
4 also applies to a recent analysis of twin
5 studies purporting to demonstrate that the
6 environmental component is an unlikely
7 explanation of both ASD risk and the increase
8 in ASD over time."

9 Now, I have a few questions
10 about that, ma'am.

11 Do you know what study they're
12 referring to?

13 If you want to flip over to
14 footnote 3, take a look at it. I think it's
15 the Taylor study, the one that they call --
16 or they identify as being a pervasive
17 problem.

18 Do you see that? That's
19 Taylor, isn't it?

20 A. I see the reference to the
21 Taylor citation.

22 Q. And you cited Taylor on page 24
23 of your report, paragraph 55, for just that
24 proposition, didn't you, the one that
25 Dr. Volk calls a problem? Page 24,

1 **paragraph 55.**

2 A. So I do cite the Taylor study
3 in my report.

4 Q. Now, Dr. Volk, as far as you
5 know, is not -- has not been paid by Johnson
6 & Johnson in this case, right?

7 MS. BROWN: Objection to the
8 form.

9 THE WITNESS: Again, I know
10 nothing about what Dr. Volk does in
11 general and whether -- what her
12 relationship is with Johnson &
13 Johnson.

14 QUESTIONS BY MR. TRACEY:

15 Q. Well, you haven't seen her
16 identified as an expert for any party in this
17 case, have you?

18 A. I'll just say in general I have
19 no knowledge of what Heather Volk does.

20 Q. Yeah.

21 Well, she says that doing what
22 you did, citing Taylor for the proposition
23 that assuming that genetic contributions
24 cannot act in concert with the environment is
25 problematic, doesn't she?

1 MS. BROWN: Objection to the
2 form. Misstates the testimony.

3 THE WITNESS: Again, she has
4 her opinion, and she's stated things,
5 certain things, within this. It is
6 the overwhelming evidence is that
7 genetics, they would agree, are an
8 important contributor to autism.

9 And I think, again, we are
10 agreeing that genetics is not
11 100 percent in terms of the risk of a
12 probability of autism.

13 Getting down into the specific
14 contributions, heritability estimates
15 from twins would still argue that the
16 overwhelming majority, however, is due
17 to genetic -- inherited genetic
18 factors.

19 QUESTIONS BY MR. TRACEY:

20 Q. Okay. Do you agree with her or
21 not?

22 A. Which statement?

23 Q. The statement we just read into
24 the record that's highlighted on the screen
25 where she identifies Taylor as a pervasive

1 **problem.**

2 A. I do not agree with that
3 statement.

4 Q. Okay. Now, Dr. Chung, it's not
5 unusual for scientists to disagree about
6 things, is it?

7 A. There can be disagreements
8 between scientists.

9 Q. I mean, two perfectly
10 competent, accomplished scientists can look
11 at the same set of data and draw different
12 conclusions, can't they?

13 MS. BROWN: Objection to the
14 form.

15 THE WITNESS: Again, although
16 two individual scientists could look
17 at the same data and for reasons come
18 to different conclusions, but over
19 time the scientific community as a
20 whole tends to look at the whole of
21 the data and come to consensus.

22 And there is consensus in the
23 scientific community that the
24 overwhelming contributors to autism
25 and ADHD are genetic factors.

1 QUESTIONS BY MR. TRACEY:

2 Q. You and I are going to explore
3 whether that's true for the rest of the day,
4 Dr. Chung.

5 But can you answer my question?

6 A. Your question, if I understood
7 it, can any two scientists disagree?

8 Q. No. I said, can two competent,
9 qualified scientists look at the same set of
10 data and draw different conclusions?

11 MS. BROWN: Objection. Asked
12 and answered.

13 THE WITNESS: Again, that's a
14 very general statement.

15 QUESTIONS BY MR. TRACEY:

16 Q. Yes, ma'am, it is.

17 A. But it is a very general
18 statement that two good scientists can have
19 different opinions about the same set of
20 data --

21 Q. We want that, don't we?

22 MS. BROWN: Wait, wait. She's
23 not done.

24 MR. TRACEY: Sorry, I thought
25 you were done. Sorry.

1 THE WITNESS: Based in part on
2 their background and their
3 understanding of the complexities of
4 the data and how one should design the
5 experiments that are necessary to come
6 to definitive conclusions.

7 QUESTIONS BY MR. TRACEY:

8 Q. You brought up a good point.
9 You ever heard the saying that
10 when you're a hammer, you think everything's
11 a nail?

12 A. I have heard of that statement.

13 Q. You're not an environmental
14 epidemiologist, are you, Dr. Chung?

15 A. I am not an environmental
16 epidemiologist.

17 Q. You've never designed an
18 environmental epidemiology study, have you?

19 A. I have not designed an
20 environmental epidemiology study.

21 Q. And you've told me before, a
22 couple times, you're not an epidemiologist?

23 A. Not an epidemiologist, but I've
24 worked with many epidemiologists over my
25 career and --

1 **Q. No doubt.**

2 A. -- and in understanding the
3 complexities of epidemiology and genetic
4 epidemiology, these areas come together in
5 terms of trying to understand cause of
6 conditions.

7 **Q. Do you know what ipse dixit**
8 **means?**

9 A. I do not.

10 **Q. Ipse dixit is a Latin term we**
11 **lawyers use, and it means because I say so.**

12 **When I was a kid --**

13 MS. BROWN: Oh, come on. Come
14 on. Come on. Come on. Please ask a
15 question.

16 MR. WATTS: Let him ask his
17 questions.

18 MS. BROWN: Yeah, but come on.

19 MR. WATTS: Object to form.

20 QUESTIONS BY MR. TRACEY:

21 **Q. When I was a kid, Dr. Chung,**
22 **and if I asked my mother if I could do**
23 **something and she'd say no, and if I asked**
24 **her why, she'd say "Because I said so."**

25 **Have you ever had that**

1 **experience?**

2 MS. BROWN: I object to the
3 form of the question.

4 THE WITNESS: I've not, but I
5 had a different mother.

6 QUESTIONS BY MR. TRACEY:

7 **Q. Okay. We don't allow that in**
8 **the law. We don't allow witnesses to say,**
9 **"Believe me because I say so."**

10 MS. BROWN: Object to the form.

11 QUESTIONS BY MR. TRACEY:

12 **Q. We require them to defend their**
13 **methodology and how they reached their**
14 **conclusions.**

15 **Does that sound fair to you?**

16 MS. BROWN: I object. It lacks
17 foundation. It's an improper
18 question.

19 THE WITNESS: I would say that
20 I use a scientific methodology in
21 which we do, as well as a scientific
22 community, strive for rigor and try to
23 understand the world around us. And
24 as we do that, we hold each other
25 accountable and to a very high

1 standard.

2 QUESTIONS BY MR. TRACEY:

3 **Q. Good point.**

4 A. And the opinion that I'm
5 rendering today is not my opinion alone. In
6 fact, it is, I would argue, the consensus of
7 the scientific community.

8 **Q. Okay. Next paragraph is**
9 **entitled "Evidence for environmental**
10 **influence on ASD risk."**

11 **Do you believe that there is**
12 **evidence for environmental influence on ASD**
13 **risk?**

14 A. I believe, as we've spoken
15 about, the heritability for autism is not
16 100 percent. Because of that, there is
17 something, if we wanted to call it
18 environment, nongenetic factors. There is
19 room for a contribution of something that is
20 not genetic.

21 **Q. They say "A large body of**
22 **evidence, including decades of research on**
23 **lead and child IQ, indicate a link between**
24 **toxic environmental exposures and poorer**
25 **developmental outcomes."**

1 **Are you familiar with the**
2 **literature she's referencing?**

3 A. Again, I think the specific
4 literature that she's referencing is the
5 association between lead.

6 **Q. Yes, ma'am.**

7 A. And between lead and
8 neurodevelopmental outcomes, postnatally, is
9 the main concern that we have in terms of
10 lead exposures.

11 **Q. They go on to say, "In animal**
12 **models and human studies, several toxic**
13 **chemicals have been implicated in ASD and**
14 **ASD-related traits and biological markers.**

15 **"Specifically, scientists have**
16 **found that air pollution exposures during**
17 **pregnancy and early infancy, at levels**
18 **typically found in large cities, are**
19 **associated with autism."**

20 **Do you agree with that, ma'am?**

21 A. Again, as we think about some
22 of these things that have been implicated
23 previously, I'll again say that we go through
24 a process iteratively of being able to have
25 more complex modeling and considering

1 multiple factors in those studies.

2 If we wanted to go back through
3 those, to my knowledge, not all of the
4 studies that are referenced here have done
5 the state-of-the-art analyses to consider all
6 those particular confounds.

7 **Q. What does that mean?**

8 A. What that means is that autism,
9 in particular, is complicated. There is not
10 one single condition that is autism. There
11 is not one single genetic or nongenetic
12 factor that is autism.

13 In trying to understand this
14 very complex human behavior, it is necessary
15 to have very complex modeling that considers
16 many, many potential confounds.

17 In doing so, these study
18 designs are extremely difficult, and having
19 the perfect study that can accurately and
20 scientifically assess the contributions of
21 these are extremely hard to do, and are still
22 getting better in their ability to answer
23 that question.

24 **Q. Do you think genetic**
25 **susceptibility contributes to smoking and**

1 **lung cancer?**

2 MS. BROWN: Objection to the
3 form. Beyond the scope.

4 THE WITNESS: Can you please
5 break that down into two separate
6 questions? Because I believe there
7 are two questions embedded within
8 that.

9 QUESTIONS BY MR. TRACEY:

10 **Q. Do you know whether there's**
11 **data that supports that people that have a**
12 **certain genetic makeup are at higher or**
13 **increased risk from smoking and lung cancer?**

14 MS. BROWN: Objection to the
15 form.

16 THE WITNESS: I have not
17 investigated that particular
18 scientific question.

19 QUESTIONS BY MR. TRACEY:

20 **Q. Do you know whether or not**
21 **Richard Doll, in 1950, had any genetic**
22 **testing to exclude the contribution of**
23 **genetic susceptibility before reaching the**
24 **conclusion he did that smoking caused lung**
25 **cancer?**

1 MS. BROWN: Objection to the
2 form.

3 THE WITNESS: To my knowledge,
4 he did not, but you bring up an
5 important point, which is that if
6 there is an exposure that has a very
7 large effect size, it can overwhelm
8 genetic factors if genetic factors are
9 not a major contributing.

10 On the other hand, if there are
11 exposures which may have a very small
12 contributing factor, it is very easy
13 for there to appear to be an
14 association where there's actually a
15 confound due to genetic factors
16 driving the signal.

17 QUESTIONS BY MR. TRACEY:

18 Q. Do you know how many diseases
19 smoking is thought to cause that are
20 generally accepted in the scientific
21 community that have effect sizes under 2?

22 A. I have not done an inventory to
23 answer that question.

24 Q. Okay. You don't know if it's 2
25 or 50?

1 A. I have not done a comprehensive
2 literature search to answer that question.

3 **Q. Do you know and understand that**
4 **smoking causes more than one disease?**

5 A. I do believe that smoking is
6 associated with more than one disease.

7 **Q. Okay. What did -- what did Sir**
8 **Bradford Hill, Austin Bradford Hill, say**
9 **about effect size in his seminal 1965 paper?**
10 **Do you know?**

11 MS. BROWN: Objection to the
12 form.

13 THE WITNESS: My understanding
14 is that the larger the effect size,
15 the more likely it is to be
16 causatively associated.

17 QUESTIONS BY MR. TRACEY:

18 **Q. Did he say that if it doesn't**
19 **have a large effect size, we just throw it in**
20 **the trash and move on?**

21 A. I believe what I said was what
22 he implied; was that if there is a large
23 effect size, it is less likely to be affected
24 by confounds.

25 **Q. I understand. That's half of**

1 **what he said, ma'am. I'm asking about the**
2 **other half.**

3 A. I would say that when there is
4 a smaller effect size, one needs to be quite
5 careful about confounding factors.

6 **Q. Fair enough.**

7 **Let's flip over to the next**
8 **page and see what these good doctors have to**
9 **say.**

10 **At the -- on the second**
11 **paragraph in the column on the left, it says,**
12 **"Some environmental factors may also reduce**
13 **the probability of autism. For example,**
14 **folate, a vitamin involved in the manufacture**
15 **and methylation of DNA, plays a critical role**
16 **in neurodevelopment."**

17 **Do you see that, ma'am?**

18 A. I see the sentence.

19 **Q. Now, you believe that's true,**
20 **don't you? You actually have that in your**
21 **PowerPoint presentations, don't you?**

22 A. I believe in terms of that
23 association, I have not seen a robust study
24 that has looked specifically at the effects
25 of folate in terms of autism that accounts

1 for the factors that need to be accounted for
2 to be able to understand that linkage.

3 **Q. Do you doubt that folate**
4 **supplementation reduces the risk of autism?**

5 A. I have no opinion one way or
6 the other. Folic, to my knowledge -- folic
7 acid, to my knowledge, is not a problem for
8 women to take during pregnancy. It's quite
9 the opposite. It protects against neural
10 tube defects. And so it is with that
11 recommendation that we recommend folic acid
12 during pregnancy for women.

13 **Q. Right.**
14 **But my question is different**
15 **than that. I appreciate that.**

16 **My question is whether you**
17 **doubt that folic acid supplements reduce the**
18 **risk of autism?**

19 A. I have no opinion. I have not
20 specifically investigated that factor.

21 I also have not seen any
22 evidence that folic acid is harmful, which is
23 why we do recommend supplementation of folic
24 acid during pregnancy.

25 **Q. Okay. You haven't reviewed the**

1 **literature on folic acid and whether it --**
2 **whether it reduces the risk of autism?**

3 MS. BROWN: I object. Asked
4 and answered.

5 THE WITNESS: At the level of
6 rigor necessary to have a
7 comprehensive opinion, I have not
8 looked at folic acid specifically for
9 association with autism in the way
10 that should be to render an opinion.

11 QUESTIONS BY MR. TRACEY:

12 Q. Okay. Down below, they've got
13 **studies of gene-environment interaction.**

14 You know what GxE means, right?

15 A. Yes, I do.

16 Q. That means gene-environment
17 **interaction, right?**

18 A. That is what scientists mean
19 when they use that term.

20 Q. They say, "As with other
21 **multifactorial conditions, it's highly likely**
22 **that the interplay of gene variants and**
23 **environmental factors contributes to a**
24 **substantial proportion of autism."**

25 Do you agree with these

1 **doctors, ma'am?**

2 A. I would say that we don't have
3 evidence in terms of specific cases to be
4 able to validate that statement.

5 Q. Okay. They say, "Highly likely
6 that the interplay between genes and the
7 environments contributes to a substantial
8 proportion of autism," ma'am, don't they?

9 A. That is the statement that they
10 make.

11 Q. Now, did you read this article
12 when it came out?

13 A. I did not.

14 Q. Okay. Do you know if anybody
15 after it was published wrote in and said,
16 "Oh, my God, Dr. Volk and Ritz, you guys are
17 clowns. That's not true"?

18 A. I don't know.

19 Q. Okay. They go on to say,
20 "Although inherited genes are fixed at
21 conception, environmental factors vary over
22 time and place and can be modified."

23 That's a true statement, isn't
24 it?

25 A. Genes can change over time and

1 that, in fact, is the underlying basis for
2 cancer in some people.

3 Q. Yes.

4 They go on to say, "Thus,
5 gene-environment interactions can provide
6 crucial biological insights and opportunities
7 for intervention at the individual, community
8 and policy levels."

9 Do you agree with that?

10 A. When it comes to autism, I
11 don't have evidence for this to be the case
12 yet.

13 Q. Okay. But this whole paper is
14 about autism, right?

15 A. This paper is purported to be
16 about autism. And, again, I don't have
17 evidence, nor do I believe they do, in terms
18 of making policy decisions that would change
19 policy in terms of the statements they make
20 in this paper.

21 Q. Okay. They go on to say, "Only
22 a handful of epidemiological studies have
23 examined the role of environmental factors in
24 combination with genetic variations and
25 autism, Table 1." We're going to look at

1 that in a second. "Early evidence is
2 striking. For example, a meta-analysis
3 revealed that a variant of the MTHFR gene,
4 which reduces the ability" -- "the body's
5 ability to convert folate in its active form,
6 contributes to higher rates of autism."

7 Do you know whether that's
8 true?

9 A. When I look in particular at
10 that manuscript, and I don't have it here
11 before me, but when I look at the date of
12 when that was published in 2012 and looking
13 at the title of this, I don't believe this
14 was a comprehensive genomic analysis. And at
15 the time when this was known, most of the
16 genetic contributors to autism were not
17 known.

18 And with a very specific
19 hypothesis that is probably not genome-wide
20 corrected for anything, I don't know that
21 that same statement would hold up with the
22 evidence that we have today.

23 Q. Well, she's making the
24 statement last year, in 2022, right?

25 A. She is referencing a paper that

1 was written and where the data were collected
2 and analyzed a long time ago.

3 Q. But she doesn't say, "But the
4 data doesn't hold up any longer," does she?

5 A. I think she is referencing a
6 paper which supports her opinion.

7 Q. Yeah.

8 She goes on to say, "Other
9 pieces of compelling evidence also underscore
10 the interaction of genetic susceptibility
11 with toxic chemicals as a major contributor
12 to autism. Researchers analyzing 206 genes
13 from an established genomics database against
14 the database that records the activities of
15 more than 10,000 chemicals on genes and gene
16 pathways found that 4,428 chemicals
17 interacted with one or more of the genes
18 linked to autism."

19 Now, do you know the paper that
20 she and her coauthors conclude is "compelling
21 evidence to underscore the interaction of
22 genetic susceptibility with toxic chemicals
23 as a major contributor to autism"?

24 It's the Carter and Blizard
25 paper.

1 A. Yes. I was just looking at the
2 reference set.

3 Yes, I am aware of this paper.

4 Q. Now, these authors, unpaid by
5 Johnson & Johnson, as far as we know, call
6 the Carter and Blizzard paper "compelling
7 evidence," don't they?

8 A. Those are these authors' words.
9 I would argue about the use of the adjective
10 "compelling."

11 Q. Okay. They go on to say,
12 "Additionally, rare genetic variants that by
13 themselves have a strong influence on ASD
14 diagnosis have been shown to have greater
15 biological perturbations when considered in
16 the presence of toxic chemical exposures in
17 in vitro studies," and then they cite two
18 papers, don't they?

19 A. They cite two papers. I've not
20 read these two papers that they cite.

21 Q. Okay. And then if you'll flip
22 over, I just want to see -- if you look at
23 Table 1 that they put on their paper, Table 1
24 is actually entitled "Epidemiologic studies
25 on specific gene-by-environment interactions

1 **in autism risk, etiologic mechanisms or**
2 **severity."**

3 **Do you see that table, ma'am?**

4 A. I do see the table.

5 **Q. And then just at the bottom,**
6 **the last reference on that page, they**
7 **reference Carter and Blizzard, don't they?**

8 A. Yes, they do.

9 (Chung Exhibit 326 marked for
10 identification.)

11 QUESTIONS BY MR. TRACEY:

12 **Q. All right. So let's turn to**
13 **Carter and Blizzard, and let's see what they**
14 **had to say.**

15 **Carter and Blizzard you have**
16 **reviewed and analyzed in your report, haven't**
17 **you?**

18 A. Yes, I have.

19 **Q. Do you know either one of those**
20 **researchers?**

21 A. I do not personally know them,
22 no.

23 **Q. Do you know their reputations**
24 **in the scientific community?**

25 A. No, I do not.

1 **Q. Have you ever talked to or**
2 **communicated with either one of them?**

3 A. I have not.

4 **Q. This is Exhibit 326.**

5 **Now, this was published in 2016**
6 **in Neurochemistry International.**

7 A. Do you know the impact factor
8 for Neurochemistry International?

9 **Q. I don't.**

10 **Does that make a difference to**
11 **you?**

12 A. To my knowledge, it's not a --
13 anyway, I would doubt it has a very high
14 impact factor.

15 **Q. Do you publish in journals**
16 **without high impact factors, Dr. Chung?**

17 A. I publish in a large number of
18 journals.

19 **Q. Yeah, but that wasn't my**
20 **question.**

21 **My question is, do you publish**
22 **in journals without high impact factors?**

23 A. With over 600 publications, I
24 publish in everything from the New England
25 Journal of Medicine, to Nature, to Science,

1 to other more specialized journals that don't
2 have as high an impact factor.

3 Q. I saw some of your publications
4 recently in something called Frontiers.

5 A. Yes.

6 Q. You know what Frontiers is,
7 don't you?

8 A. Yes.

9 Q. Frontiers is sometimes referred
10 to as a predatory journal, isn't it?

11 MS. BROWN: Objection to the
12 form of the question.

13 THE WITNESS: I don't believe I
14 was the senior or first author for any
15 publication in Frontiers.

16 QUESTIONS BY MR. TRACEY:

17 Q. Well, your name is on the
18 paper?

19 A. My name is on many papers.

20 Q. It is, many.

21 You know that Frontiers is
22 called a predatory journal, right?

23 MS. BROWN: Asked and answered.

24 I object.

25 THE WITNESS: I did not

1 know that -- I don't even know what a
2 predatory journal is, but, no, I did
3 not know that.

4 QUESTIONS BY MR. TRACEY:

5 Q. Okay. Well, you brought up the
6 subject of impact factor, right?

7 A. Yes.

8 Q. Are you trying to diminish the
9 journal Neurochemistry International? Are
10 you trying to imply that we ought not pay
11 attention to anything published in that?

12 MS. BROWN: Objection to the
13 form. Assumes testimony.

14 THE WITNESS: I asked a simple
15 question because you seemed to be
16 concerned about the impact of certain
17 journals as you've gone through your
18 line of questioning.

19 QUESTIONS BY MR. TRACEY:

20 Q. Okay. Should we assess the
21 science on the science or on the journal in
22 which it's contained?

23 MS. BROWN: Objection to the
24 form.

25 THE WITNESS: I think we should

1 assess the science on the science, but
2 many times you will see scientists who
3 will submit their manuscript to one
4 journal and not have it accepted
5 because of issues with the science
6 that they have submitted.

7 QUESTIONS BY MR. TRACEY:

8 **Q. Certainly happened to you,**
9 **hasn't it?**

10 A. It's happened to every good
11 researcher that's submitted.

12 **Q. Yeah. Nothing wrong with that,**
13 **is there?**

14 A. There can be.

15 **Q. Sure, there can be.**

16 A. But there may not be.

17 **Q. But by itself, that's not an**
18 **indication of science that ought not be paid**
19 **attention to, right?**

20 A. I think one has to look at the
21 individual case, but in general, I will just
22 say that Neurochemistry International is not
23 a journal that many people commonly would
24 regard as a high impact factor journal.

25 **Q. Well, it's got an impact --**

1 they -- somebody just handed me a note. It's
2 got an impact factor of 4.3.

3 A. Okay.

4 Q. That's pretty good, isn't?

5 A. It's not above 10.

6 Q. It's not, but it's pretty good.

7 Your average journal is what,
8 under 3, under 2 and a half?

9 A. As we think about journals with
10 higher impact, we use a threshold of 10.

11 Q. All right. If you have
12 published in a journal, Dr. Chung, with an
13 impact factor of under 10, should we just
14 take all of that literature that you publish
15 and throw it in the trash?

16 A. No. We should judge it on its
17 merits.

18 Q. Okay. That's the right thing
19 to do, isn't it?

20 A. It is always the right thing to
21 do, to judge the data and the science on its
22 merits.

23 Q. So let's see what these doctors
24 that Dr. Volk referenced had to say.

25 The name of this study is

1 **"Autism genes are selectively targeted by**
2 **environmental pollutants, including**
3 **pesticides, heavy metals, bisphenol A,**
4 **phthalates and many others in food, cosmetics**
5 **or household products."**

6 **And they say in the abstract,**
7 **"The increasing incidence of autism suggests**
8 **a major environmental influence."**

9 **You disagree with that, do you?**

10 A. I would say the next sentence
11 is critical. "Epidemiology has implicated
12 many candidate genes and genetics -- in
13 genetics, many susceptibility genes."

14 Q. **Okay. You want to take those**
15 **sentences together then?**

16 A. I'd focus on the second
17 sentence.

18 Q. **But I want to focus on the**
19 **first.**

20 **How come you don't want to**
21 **focus on the first that says "major**
22 **environmental influences"? Is that because**
23 **that doesn't support your opinion?**

24 MS. BROWN: Objection.

25 Argumentative.

1 THE WITNESS: Again, my opinion
2 is based on the data, and the
3 overwhelming data is reliable in terms
4 of genetic contributions.

5 QUESTIONS BY MR. TRACEY:

6 Q. Well, this -- these authors
7 apparently disagree with you, correct?

8 A. These authors are focusing on
9 environmental pollutants.

10 Q. And genes?

11 A. And I have -- and they do
12 consider genes.

13 Q. And they also consider drugs,
14 including acetaminophen, in this paper, don't
15 they?

16 A. Yes, and they consider
17 acetaminophen.

18 Q. All right. So they say -- they
19 go on to say, "Epidemiology has implicated
20 many candidates and genetics, many
21 susceptibility genes. Genes/environmental
22 interactions in autism were analyzed using
23 206 autism susceptibility genes from the
24 Autworks database to interrogate about
25 1 million gene -- chemical/gene interactions

1 in the Comparative Toxicogenomics Database.

2 "Any bias towards autism
3 susceptibility genes, ASGs, was statistically
4 determined for each chemical. Many major
5 compounds identified in epidemiology,
6 including," and then they list a whole list
7 of them, including acetaminophen.

8 Do you see that?

9 A. I see that, but I don't want to
10 dismiss what you have correctly pointed out,
11 which is that they do a very, very large
12 number of tests statistically, when I think
13 about the statistical penalty.

14 So they're looking at millions
15 of different independent tests in terms of
16 doing this and --

17 Q. Yes.

18 A. -- to my understanding of what
19 they've done, they have not statistically
20 penalized themselves for those multiple
21 independent comparisons.

22 Q. You think they didn't do that?

23 A. I don't believe they have
24 rigorously done this to be able to take into
25 account by chance alone some possible

1 associations.

2 Q. Okay. Well, flip over and
3 let's see what they say about acetaminophen
4 and other chemicals that are known causes of
5 autism. Okay?

6 In the introduction, about
7 two-thirds of the way down, it says, "Certain
8 drugs used in pregnancy, including
9 valproate" --

10 A. Can you just hold on? I just
11 want to --

12 Q. Sorry. "Certain drugs used in
13 pregnancy, including valproate."

14 Let's stop there.

15 MS. BROWN: Hang on a second.
16 Can she just have a moment to find it
17 on her copy?

18 In think you're in the first
19 paragraph of the introduction; is that
20 right?

21 MR. TRACEY: Yes, ma'am.

22 MS. BROWN: Okay.

23 THE WITNESS: Okay.

24 MS. BROWN: Just take a -- take
25 as long as you need to be able to

1 answer the question.

2 QUESTIONS BY MR. TRACEY:

3 **Q. Doc, just -- that sentence**
4 **that's highlighted, "Certain drugs used in**
5 **pregnancy, including valproate."**

6 **You know what valproate is?**

7 A. Yes.

8 **Q. One of the brand names for**
9 **valproate is Depakote, right?**

10 A. Yes.

11 **Q. Does Depakote -- does Depakote**
12 **cause autism?**

13 A. To be able to explain the
14 detail, there are certain exposures that have
15 been -- I'm going to use the term --
16 associated with an outcome such as autism.

17 In terms of causation, looking
18 for potential confounds for those, have not
19 been completely and adequately done for each
20 of the different exposures that they list.

21 **Q. What's the label for Depakote**
22 **say about whether it causes autism? Do you**
23 **know?**

24 A. I don't know the label.

25 **Q. We'll look at that in a few**

1 **minutes and see what the manufacturer of the**
2 **drug says.**

3 A. Okay.

4 Q. Would that be helpful to you?

5 A. To my knowledge, valproic acid
6 is not what we're talking about, but...

7 Q. Do you know what analogy is in
8 the context of the Bradford Hill criteria?

9 A. I have a general understanding.

10 Q. Okay. After valproate, they
11 list SSRIs and some other drugs, and then
12 they reference acetaminophen again, right?

13 A. They do reference
14 acetaminophen.

15 Q. And what they do, is they say,
16 **"These and other environmental risk factors**
17 **are referenced in Table 1."**

18 Do you know what a risk factor
19 **is?**

20 MS. BROWN: Objection to the
21 form.

22 THE WITNESS: Again, these are
23 their words for what they're
24 considering risk factor, but it's
25 implied to be something that increases

1 the probability of the outcome.

2 QUESTIONS BY MR. TRACEY:

3 Q. Yeah. That's a generally
4 accepted and understood term in science,
5 isn't it?

6 A. I believe so.

7 Q. And what you want to do in
8 science is identify risk factors that are
9 modifiable; that is, risk factors that we
10 can -- we can intervene in and reduce the
11 risk, right?

12 MS. BROWN: Objection to the
13 form.

14 THE WITNESS: I think there are
15 different reasons that we do science,
16 but, yes, one is to be able to
17 minimize the burden of a condition.

18 QUESTIONS BY MR. TRACEY:

19 Q. And then if we turn to Table 1,
20 they have this -- it's called "compounds that
21 have been implicated in autism and
22 epidemiological studies or where different
23 blood, hair or tissue levels have been
24 reported. Where available, relevant animal
25 studies are noted."

1 **That's the name of the table,**
2 **right?**

3 A. That is the title of the table.

4 **Q. And just flip down on the first**
5 **page, you've got chlorpyrifos.**

6 **Do you know what that is?**

7 A. Can you highlight where you're
8 showing that?

9 **Q. It's the second from the last**
10 **one.**

11 A. Yes, it's under --

12 **Q. Chlorpyrifos.**

13 A. Under the category of
14 pesticides.

15 **Q. Yes, ma'am.**

16 **Do you know what that is?**

17 A. I assume it's a pesticide.

18 **Q. Yes, ma'am.**

19 **Do you know whether or not it**
20 **causes autism?**

21 A. Within this -- as I said, I'll
22 go back to this, that many epidemiological
23 studies that have been done to be able to
24 look at compounds, but they have not all been
25 done with the level of rigor to look for all

1 the potential confounds.

2 Q. Do you -- do you know, ma'am,
3 whether or not it's generally accepted that
4 chlorpyrifos causes autism or contributes to
5 cause autism?

6 A. Again, when one looks at the
7 timestamp in terms of when these studies were
8 done, these studies did not include all the
9 potential confounds that can now be done to
10 rigorously assess any potential environmental
11 exposure or contributor to autism.

12 Q. Can you answer my question?

13 MS. BROWN: Objection to the
14 form.

15 THE WITNESS: I'm sorry, can
16 you repeat the question?

17 QUESTIONS BY MR. TRACEY:

18 Q. Yeah.

19 Do you know whether it's
20 generally understood in the medical and
21 scientific community that chlorpyrifos can
22 contribute to cause autism?

23 A. I thought I answered that
24 question, which is -- but to be clearer --

25 Q. You didn't, ma'am.

1 MS. BROWN: Well, let's let
2 her --

3 QUESTIONS BY MR. TRACEY:

4 Q. What you did is you told me a
5 lot of things that hasn't been done, that
6 there's timestamps, and I don't know if a
7 bunch of work's been done.

8 I want to know whether you know
9 here and now whether it's generally
10 understood in the medical and scientific
11 community that chlorpyrifos contributes to
12 cause autism.

13 MS. BROWN: I object to the
14 speech, and she's asked and answered
15 that question.

16 And she was in the middle of
17 re-answering it when you interrupted
18 her, so I --

19 MR. TRACEY: Yeah. I don't
20 want another nonresponsive answer.

21 MS. BROWN: Let -- no. No.
22 You asked a question. She gets to
23 answer it. Let's give her a moment to
24 answer the question.

25

1 QUESTIONS BY MR. TRACEY:

2 Q. Can you answer my question,
3 Dr. Chung?

4 A. For simplicity, no, I don't
5 believe it's generally accepted in terms of
6 this being an exposure in terms of autism.

7 Q. Okay.

8 A. So the simple answer, no.

9 Q. That's all I'm looking for is
10 the simple answers.

11 If you flip over to page 87,
12 Table 1 continues -- oh, no, sorry, 86,
13 you'll see lead, manganese and mercury there
14 under heavy metals.

15 Do you know what -- ma'am,
16 whether lead, manganese and mercury are
17 associated with autism?

18 A. I won't comment on each of
19 these, but certainly lead is something that
20 we're concerned about in terms of children
21 and exposure to lead.

22 Q. Okay. Under herbicide --
23 sorry. On the next page, you'll see -- oh,
24 sorry. I'm getting ahead of myself with
25 the -- sorry, Mike.

1 **Yeah, there's lead, mercury and**
2 **manganese.**

3 **And if you go to the next page,**
4 **you'll see air pollution.**

5 **Do you remember looking at**
6 **air pollution on the national -- the**
7 **Environmental Health Science website where**
8 **they specifically talked about a specific**
9 **gene being associated with autism and**
10 **air pollution?**

11 A. I do remember where they pulled
12 out one specific gene. Air pollution is
13 complex because it's often related to
14 particular location, and those locations can
15 be included or can include complex other
16 socioeconomic and social determinants of
17 health that are related to air pollution.

18 So it's a complex set of
19 questions that get rolled up into
20 air pollution.

21 Q. Okay. Let's keep flipping
22 until we get to 89 because there's one called
23 drugs used in pregnancy or to induce or delay
24 labor.

25 And the number one on the list

1 **there is acetaminophen, isn't it?**

2 A. I see acetaminophen listed in
3 this table, among -- with many, many, many
4 other things.

5 Q. Yeah, we do.

6 **But there it is, right?**

7 A. I see it listed amongst many
8 other things, yes.

9 Q. Why do you feel the need to add
10 **things all the time when I ask questions?**

11 MS. BROWN: I object. That's
12 argumentative.

13 She's answering your questions
14 truthfully and accurately and
15 completely, and I would expect that's
16 what you want from her to do.

17 QUESTIONS BY MR. TRACEY:

18 Q. Ma'am, why do you feel the need
19 **to add things?**

20 MS. BROWN: No, I object.

21 Don't answer that question.

22 It's argumentative.

23 QUESTIONS BY MR. TRACEY:

24 Q. Do you feel like you're an
25 **advocate here, ma'am, or are you a witness?**

1 A. I feel like I need to represent
2 the scientific information as accurately as
3 possible.

4 Q. Okay. And you think in order
5 to do that, you need to add things to my
6 questions?

7 MS. BROWN: Objection to the
8 form. Argumentative.

9 THE WITNESS: I think in terms
10 of representing this, I'm trying to
11 represent the entirety of the
12 scientific literature and community.

13 QUESTIONS BY MR. TRACEY:

14 Q. All right. Flip over to the
15 next page, because on the next page, we have
16 thalidomide and valproate, don't we? That's
17 also under Table 1.

18 A. Yes, those are both listed.

19 Q. Pardon me?

20 A. Yes, thalidomide, valproate are
21 both listed.

22 Q. And then they have other drugs,
23 and they have cannabis, don't we -- don't
24 they? That's marijuana, right?

25 A. They do have that listed.

1 **Q. Is marijuana associated with**
2 **neurodevelopmental disorders, including**
3 **autism and ADHD?**

4 A. Again, this is a complicated
5 analysis that can be confounded by who would
6 use any of the medications that are on this
7 list, and I think that has to be considered
8 as one designs these studies to ask that
9 question.

10 **Q. Can you not answer the question**
11 **whether smoking pot during pregnancy is**
12 **harmful from a neurotoxic viewpoint to**
13 **children?**

14 A. I've not investigated that
15 particular question.

16 **Q. Do you know what the American**
17 **College of Obstetricians and Surgeons -- or**
18 **Gynecologists says about whether or not it's**
19 **okay to smoke pot while you're pregnant?**

20 A. I would assume that in general,
21 they would say that for anything, any
22 medication, that you should talk with your
23 doctor, and you should be able to discuss
24 that with them.

25 **Q. Well, let's just a general**

1 **comment about everything, isn't it?**

2 A. That is a general comment about
3 during pregnancy, one should talk to their
4 doctors, and one should be able to consider
5 the context of that individual person.

6 Q. But my question was whether
7 you're aware of what ACOG says about whether
8 smoking pot while you're pregnant leads to
9 neurodevelopmental -- poor neurodevelopmental
10 outcomes includes autism and ADHD.

11 A. I'm not aware of a specific
12 policy statement.

13 Q. Do you know whether or not
14 acetaminophen works on the endocannabinoid
15 system?

16 A. Exact -- explain what you mean
17 by your question.

18 Q. Do you know whether it has an
19 effect on the endocannabinoid system in
20 humans?

21 A. Can you define for me what you
22 mean by the "endocannabinoid system"?

23 Q. No.

24 Can you not answer it just
25 generally?

1 A. I can't answer your question
2 then, I'm sorry.

3 **Q. Okay. Do you ever talk to**
4 **women about whether it's a good idea to smoke**
5 **pot during pregnancy?**

6 A. I'm not an obstetrician.

7 **Q. Okay. Do you -- by the way,**
8 **when's the last time you diagnosed a child in**
9 **a clinic with autism?**

10 A. I am not a clinical
11 psychologist that performs an ADOS and an ADI
12 myself to make those diagnoses. I'm a
13 geneticist who makes genetic diagnoses, often
14 which for conditions that are associated with
15 autism, but I use a team of behaviorists who
16 make a behavioral diagnosis of autism.

17 **Q. So the answer to my question**
18 **is, you don't make autism diagnoses, correct?**

19 MS. BROWN: Objection. Asked
20 and answered.

21 THE WITNESS: I make genetic
22 diagnoses.

23 QUESTIONS BY MR. TRACEY:

24 **Q. But autism is not a genetic**
25 **diagnosis, ma'am. It's a behavioral,**

1 **clinical diagnosis, correct?**

2 MS. BROWN: Objection to the
3 form.

4 THE WITNESS: That is correct.
5 And as I stated, there are individuals
6 who are reliable in performing
7 measures to make a diagnosis of
8 autism, things like the ADOS and the
9 ADI, and they are trained specifically
10 to do so.

11 QUESTIONS BY MR. TRACEY:

12 **Q. Right.**

13 **But if we're going to be clear**
14 **about what you do, you do not yourself**
15 **diagnose children with autism?**

16 A. I do not diagnose children with
17 autism, but I have decades of experience
18 caring for individuals with autism. I'm very
19 familiar with the signs and symptoms and how
20 to support children. But my formal part of a
21 multi-disciplinary team is not to make that
22 diagnosis but to be able to do many other
23 things to support those individuals.

24 **Q. Okay. Have you ever diagnosed**
25 **a child with autism in your career?**

1 A. I certainly recognize the signs
2 and symptoms. I don't formally give that
3 diagnosis. Again, there is another member of
4 our multi-disciplinary team who makes that
5 formal diagnosis, but I'm able to recognize
6 the signs and symptoms.

7 **Q. Okay. But the answer to my**
8 **question is, you have never in your career**
9 **formally diagnosed a child with autism?**

10 A. Again, that role is for someone
11 who is a behavioral psychologist, usually,
12 and to be able to make that diagnosis, and
13 that is not my official training. But my
14 years of experience in clinical practice have
15 taught me, by experience, how to recognize
16 the signs and symptoms and how to support
17 those individuals.

18 **Q. Okay. Let's flip over to**
19 **page 91 of Carter and Blizzard.**

20 **This is under the Results**
21 **section.**

22 **And it's -- the 3.1 says,**
23 **"Genes affected by compounds implicated in**
24 **autism."**

25 **And they start off, "Of the**

1 **named pollutants implicated in autism, 45**
2 **showed enrichment values, the p-value less**
3 **than .05," right?**

4 **Are you --**

5 A. Sorry, I'm trying to find --

6 MS. BROWN: I think you --

7 let's just find it on her hard copy.

8 It looks like Section 3.1.

9 QUESTIONS BY MR. TRACEY:

10 Q. **3.1, page 91 at the top under**
11 **Results.**

12 A. Okay. Do you mind going back?

13 Sorry.

14 Q. **Yeah, sorry.**

15 It says, "3.1. Genes affected
16 **by compounds implicated in autism, Figure 1."**

17 And we'll get to Figure 1.

18 And then it talks about the

19 **named pollutants first which showed**

20 **enrichment values at less than .05.**

21 Do you see that?

22 A. I see that statement.

23 Q. **Okay. And then they list a**
24 **bunch of -- a bunch of compounds with the**
25 **most significant enrichment scores.**

1 And then two paragraphs down,
2 they talk about drugs, they're switching
3 topics, and they say, "Drugs with the most
4 significant enrichment scores included," they
5 got SSRI, antidepressants, thalidomide; drugs
6 of abuse - cocaine, methamphetamine, ethanol,
7 it's alcohol, et cetera, et cetera. And then
8 down there at the bottom, between -- or after
9 thalidomide you see acetaminophen as being
10 one of the drugs with the most significant
11 enrichment score.

12 Right?

13 A. I see -- I see what you're
14 reading, yes.

15 Q. And then if we flip over to --
16 and then they cite us to Figure 2,
17 somewhere -- there we go, Figure 2. And then
18 let's flip over to Figure 2, and I want to
19 look at the drugs with the most significant
20 enrichment scores on Figure 2.

21 Now, number 1, the highest
22 enrichment score, showing the highest
23 chemical autism susceptibility gene
24 interaction, was valproic acid at 130, wasn't
25 it?

1 MS. BROWN: Object to form.

2 THE WITNESS: So, again, based
3 on this, this is not based on human
4 epidemiological data, just so that
5 we're clear on this in terms of
6 implicating any of these exposures and
7 what we see in actual people.

8 QUESTIONS BY MR. TRACEY:

9 Q. Okay. But I didn't ask you any
10 of that.

11 Did I, ma'am?

12 A. So I'm just saying what I'm
13 concerned about are these exposures in actual
14 people and the behaviors that we see in
15 actual people, and none of the data from this
16 paper are implicating exactly what I think
17 we're talking about today.

18 Q. Okay. Well, what we're talking
19 about is whether or not genes, autism
20 susceptibility genes, are enriched when
21 they're exposed to chemicals, right?

22 MS. BROWN: Objection to the
23 form. Vague.

24 THE WITNESS: Again, these are
25 not experiments, for instance, that

1 were done in human people in looking
2 at gene expression in human brains and
3 being able to understand these
4 exposures and how that relates to the
5 behaviors associated with those
6 individuals.

7 So these are many steps removed
8 from the actual biological question.

9 QUESTIONS BY MR. TRACEY:

10 **Q. We're going to get there.**

11 **We're going to get there. Trust me.**

12 **But these are gene enrichment**
13 **scores. Do you not use gene enrichment**
14 **scores in your own work?**

15 A. Not in the way that these are
16 done, and I'm not clear that this biology
17 that they're trying to underscore is actually
18 relevant to the human biology that we see.

19 **Q. Well, can you answer my**
20 **question, though? Isn't number 1 on the list**
21 **valproic acid? That's the number 1**
22 **enrichment score, isn't it?**

23 MS. BROWN: Objection to the
24 form. Misstates the document.

25 THE WITNESS: For this

1 experiment that they have performed,
2 yes, valproic acid is the one that has
3 the strongest effect.

4 Whether or not that's relevant
5 to the human biology, that's what I'm
6 saying there's a gap.

7 QUESTIONS BY MR. TRACEY:

8 Q. Okay. In a few minutes, ma'am,
9 we're going to look at the epidemiology of
10 valproic acid and what it says about autism
11 to see if it's coherent with what Carter and
12 Blizard show here.

13 Is that fair?

14 A. If you'd like to, we certainly
15 can.

16 Q. That's a fair thing to do,
17 isn't it?

18 A. We can certainly do that later
19 today.

20 Q. Okay. So I've got that
21 highlighted.

22 And then number 2 on the list,
23 the chemical compound, the drug that enriches
24 the second-most autism susceptibility genes
25 is acetaminophen, isn't it?

1 A. I see within that figure, yes,
2 acetaminophen is the number 2.

3 **Q. Number 1 and number 2, right?**

4 A. Valproic acid being number 1
5 and acetaminophen being number 2.

6 **Q. And thalidomide is a distant**
7 **third in that section of the graph, isn't it?**

8 A. Again, I don't know how to
9 interpret these data to the human experience
10 of autism and the prenatal exposure. I don't
11 believe these are a direct correlation or a
12 causation.

13 **Q. Perhaps I can help you with**
14 **that later, but can you answer my question**
15 **about whether or not thalidomide is a distant**
16 **third after valproic acid and acetaminophen?**

17 A. Within this particular cluster
18 that you're showing on the right, within that
19 quartet of those four exposures, yes, that is
20 number 3.

21 **Q. Do you know whether or not, as**
22 **we sit here today, it is generally accepted**
23 **that thalidomide causes autism?**

24 MS. BROWN: Objection. Asked
25 and answered.

1 THE WITNESS: Again, I have not
2 done a comprehensive review --

3 QUESTIONS BY MR. TRACEY:

4 Q. Oh, I -- I'm sorry, you did
5 answer that. You're right. Sustained.

6 MS. BROWN: Thank you, Judge.
7 I appreciate it.

8 MR. TRACEY: Yeah. Yeah, I
9 forgot. My apologies. You did answer
10 that. Okay. All right. We're going
11 to look at Depakote in just a few
12 minutes.

13 MS. BROWN: Should we take a
14 break?

15 MR. TRACEY: Yeah, yeah. We're
16 going to do -- yeah, go ahead. Sorry.

17 VIDEOGRAPHER: The time is
18 11:28 a.m., and we're off the record.

19 (Off the record at 11:28 a.m.)

20 VIDEOGRAPHER: The time is
21 11:41 a.m., and we're on the record.

22 (Chung Exhibit 329 marked for
23 identification.)

24 MR. TRACEY: Do you have that,
25 Danny?

1 DANIEL OLIVO: Yeah.

2 QUESTIONS BY MR. TRACEY:

3 Q. Doctor, I'm going to hand you
4 the Exhibit 329 here in a second, and it's
5 the Depakote label.

6 Generic name of Depakote is
7 valproic acid, right?

8 A. Yes.

9 Q. And just as sort of a threshold
10 matter, Depakote is an antiseizure medicine,
11 and it's also used for prevention of
12 migraines.

13 Is that your understanding?

14 A. Yes, that's my understanding.

15 Q. It's not indicated for minor
16 aches and pains, right?

17 A. Not to my knowledge.

18 Q. Okay. It's got some black box
19 warnings up there on that first page.

20 The first one is
21 hepatotoxicity.

22 Do you see that?

23 A. I see that.

24 Q. And then, just the way these
25 new and improved labels work, if you flip

1 over to the fourth page, it's got Use in
2 Specific Populations, and Pregnancy is 8.1.
3 It's the same in every label, as you probably
4 know. You always go to 8.1 in a label to
5 look at pregnancy.

6 And so we're going to do that.
7 We're going to flip over to page 30, just to
8 show you that we're in the pregnancy section.
9 And then on the next page, because it starts,
10 actually, on the next page, 31, they say
11 here -- under the -- it's the -- I don't
12 know, fourth or fifth paragraph from the
13 bottom under Risk Summary. It says,
14 "Epidemiological studies have indicated that
15 children exposed to valproate in utero have
16 lower IQ scores and higher risk of
17 neurodevelopmental disorders compared to
18 children exposed to another AED," which is
19 antiepileptic drug, "in utero or to no AEDs,"
20 and then they say, "See warnings."

21 And then just below that it
22 says, "An observational study has suggested
23 that exposure to valproate products during
24 pregnancy increases the risk of autism
25 spectrum disorders."

1 And then they go on to say, "In
2 animal studies, valproate administration
3 during pregnancy resulted in fetal structural
4 malformations similar to those seen in humans
5 and neurobehavioral deficits in the offspring
6 at clinically relevant doses."

7 And so that is -- those are
8 sort of the three things I want to talk about
9 in this label, which is under the Risk
10 Summary.

11 Okay?

12 A. Okay.

13 Q. Now, if you flip over two more
14 pages, we finally get to the data section.
15 And you'll see it's the paragraph that -- it
16 says -- it starts off, "Effect on IQ and
17 neurodevelopmental effects."

18 And then the second paragraph
19 down is the neurodevelopmental effect
20 paragraph.

21 Okay? Are you oriented to
22 where we are?

23 A. I see that.

24 Q. So what this FDA -- you know
25 this is an FDA-approved label, right?

1 MS. BROWN: Objection. Lacks
2 foundation.

3 QUESTIONS BY MR. TRACEY:

4 Q. Or do you know that?

5 A. I know this is an FDA-approved
6 medication.

7 Q. Okay. They say, "Although the
8 available studies have methodological
9 limitations, the weight of the evidence
10 supports a causal association between
11 valproate exposure in utero and subsequent
12 adverse effects on neurodevelopment,
13 including increases in autism spectrum
14 disorders and attention-deficit/hyperactivity
15 disorder. An observational study has
16 suggested that exposure to valproate products
17 during pregnancy increases the risk of autism
18 spectrum disorders. In this study, children
19 born to mothers who had used valproate
20 products during pregnancy had 2 point
21 times" -- "2.9 times the risk of developing
22 autism spectrum disorders compared to
23 children born to mothers not exposed to
24 valproate products during pregnancy."

25 I'm going to stop there for a

1 **second because that's a big mouthful.**

2 **Were you aware, ma'am, that**
3 **this warning in the Depakote label about the**
4 **risk of autism and that the weight of the**
5 **evidence supporting a causal association was**
6 **based on one observational epidemiology**
7 **study?**

8 MS. BROWN: I object. It lacks
9 foundation and assumes facts.

10 THE WITNESS: I'm not an expert
11 in labeling, and I hadn't reviewed
12 this document prior to today.

13 QUESTIONS BY MR. TRACEY:

14 **Q. Okay. You see that in this**
15 **label, this document, at least as far as I**
16 **can tell, they have only referenced one**
17 **study, and I believe the record is going to**
18 **reflect it's one of the Christensen studies.**

19 **Are you familiar with**
20 **Dr. Christensen and his work?**

21 MS. BROWN: Object to the first
22 part of the question as lacking
23 foundation.

24 THE WITNESS: I haven't
25 comprehensively reviewed the valproic

1 acid data and relationships to autism
2 or ADHD in preparation for today.

3 QUESTIONS BY MR. TRACEY:

4 Q. Okay. Have you ever
5 comprehensively reviewed it?

6 A. Not in the modern era.

7 Q. What's the modern era for
8 Dr. Chung?

9 A. Within the last five years.

10 Q. Okay. They say below that,
11 "Another observational study found that
12 children who were exposed to valproate in
13 utero had an increased risk of ADHD of 1.48
14 compared with the unexposed children.
15 Because these studies were observational in
16 nature, conclusions regarding a causal
17 association between in utero valproate
18 exposure and an increased risk of autism
19 spectrum disorder and ADHD cannot be
20 considered definitive."

21 Did I read that mostly correct?

22 A. Yes, I believe you did.

23 Q. Now, is it -- it is true,
24 because I've read it in your report, that you
25 believe that all observational studies have

1 **methodological flaws, right?**

2 A. I believe that we're getting
3 better over time, but there are limitations
4 with epidemiological observational studies.

5 Q. Do you know whether either one
6 of the observational studies that, candidly,
7 the label says have methodological flaws,
8 whether either one of them accounted for
9 confounding by genetics?

10 MS. BROWN: I object.

11 Are you talking about the two
12 studies referenced in this label?

13 MR. TRACEY: I am.

14 MS. BROWN: Okay. I object.
15 Lacking foundation. Calls for
16 speculation.

17 THE WITNESS: Again, I haven't
18 reviewed these papers in
19 preparation -- or in preparation of my
20 opinion or for today.

21 QUESTIONS BY MR. TRACEY:

22 Q. Would it surprise you to know,
23 Dr. Chung, that neither one of the
24 observational studies referenced in this
25 label accounted for any genetic confounding?

1 MS. BROWN: Objection. Assumes
2 facts. Lacks foundation.

3 THE WITNESS: Again, I don't
4 know the studies, but it would not
5 surprise me if they were things that
6 were not done within the last year or
7 two.

8 QUESTIONS BY MR. TRACEY:

9 Q. You're not of the opinion,
10 Dr. Chung, are you, that until and unless
11 observational epidemiology studies account
12 for potential genetic confounders using the
13 latest and greatest technology that we can't
14 reach any conclusions about observational
15 studies, are you?

16 MS. BROWN: Objection. Vague.

17 THE WITNESS: Again, I think
18 one looks at the relative risk
19 associated with these, understanding
20 that is something that one considers
21 in terms of what that risk is. If
22 something were associated with a
23 200-fold increased risk, I think it
24 would be more obvious that there were
25 unlikely to be confounds accounting

1 for that signal.

2 When you see signals that are
3 associated with a relative risk that
4 are more modest, 1 to 2, for instance,
5 I think it gets more complicated and
6 one has to be able to consider the
7 confounds.

8 Again, valproic acid was not
9 something that I was asked to comment
10 on, and I did not prepare an opinion
11 on valproic acid.

12 QUESTIONS BY MR. TRACEY:

13 **Q. Well, the relative risk of**
14 **ADHD, according to this label, are modest by**
15 **your standards, 1.48, right?**

16 MS. BROWN: Objection.

17 Misstates the label.

18 THE WITNESS: Again, I was not
19 considering the literature for
20 valproic acid, and I don't have an
21 opinion to render on valproic acid.

22 QUESTIONS BY MR. TRACEY:

23 **Q. I didn't ask you about that. I**
24 **asked you about the number.**

25 **You said modest numbers are**

1 **between 1 and 2, which is exactly what we see**
2 **in this label, right, on ADHD?**

3 MS. BROWN: Objection.

4 THE WITNESS: I will say that
5 what I see and on the screen before me
6 is a hazard ratio of 1.48.

7 QUESTIONS BY MR. TRACEY:

8 **Q. And then on autism, we see a**
9 **2.9 relative risk, right?**

10 A. Yes, that's what I see above,
11 is 2.9.

12 **Q. Now, is this label and the**
13 **language that they use -- scroll up a little**
14 **bit -- that the weight of the evidence --**
15 **"Although studies have methodological**
16 **limitations, the weight of the evidence**
17 **supports a causal association between**
18 **valproate exposure in utero and subsequent**
19 **adverse effects on neurodevelopment,**
20 **including ASD and ADHD," is that statement**
21 **consistent with the enrichment score that**
22 **Carter and Blizzard found in their**
23 **gene-chemical interaction study?**

24 MS. BROWN: Objection. Lacks
25 foundation.

1 THE WITNESS: Again, as we've
2 talked about within the Carter study,
3 that's not based on the actual biology
4 in humans, and I think that needs to
5 be looked at separately from anything
6 that's being referenced here in terms
7 of epidemiology studies in people.

8 QUESTIONS BY MR. TRACEY:

9 Q. Okay. But is it consistent
10 with what this epidemiology shows, which is a
11 human study, right?

12 MS. BROWN: Asked and answered.
13 I object.

14 THE WITNESS: Again, these are
15 two small data points, but it's not
16 taking into consideration the totality
17 of the data that Carter looked at.

18 QUESTIONS BY MR. TRACEY:

19 Q. I'm not -- I'm not following
20 you.

21 Carter did an enrichment score
22 for valproic acid, right? And it had the
23 highest enrichment score when it interacted
24 with autism susceptibility genes, of drugs,
25 right?

1 A. So all I'm saying is that
2 you've cherry-picked two data points from the
3 Carter study. They've looked at thousands of
4 compounds in terms of doing that, and I
5 haven't seen an association, in terms of what
6 you're describing to me, looking at
7 epidemiological data with all of the
8 different things that they looked at, to know
9 whether that's a consistent pattern to assess
10 the validity of the method that they used.

11 Q. Okay. But I'm not asking you
12 about any of that, Doctor.

13 What I'm asking is very simple.
14 Is the conclusions that they reached in this
15 label about the weight of the evidence
16 supporting a causal association consistent
17 with Carter and Blizzard's high enrichment
18 score?

19 MS. BROWN: Objection. Asked
20 and answered.

21 You can answer it again.

22 THE WITNESS: Again, these are
23 two of thousands of data points, and
24 by chance --

25

1 QUESTIONS BY MR. TRACEY:

2 Q. But I'm only asking you about
3 one.

4 MS. BROWN: Let her finish,
5 please.

6 QUESTIONS BY MR. TRACEY:

7 Q. I'm only asking you about one.

8 MS. BROWN: She's answering
9 your question.

10 Go ahead.

11 MR. TRACEY: No. No, she's
12 not. She's answering about a thousand
13 other things.

14 MS. BROWN: That's the answer
15 to the question.

16 MR. TRACEY: You know -- you
17 know what I'm asking, Doctor.

18 MS. BROWN: She's giving you
19 the answer.

20 THE WITNESS: What I'm saying
21 is that by chance alone, one could
22 have seen this same association that
23 you're seeing. And scientifically and
24 rigorously to assess whether or not
25 that would be consistent, I've told

1 you the methodology that we would use
2 to be able to understand that within
3 the larger context of the studies that
4 were done.

5 QUESTIONS BY MR. TRACEY:

6 **Q. But the confidence intervals in**
7 **these studies exclude chance, don't they?**

8 A. That's a different issue that
9 you're raising so --

10 **Q. Well, you raised the issue of**
11 **chance, I thought.**

12 **You were talking about Carter**
13 **and Blizzard?**

14 A. So talking about Carter and
15 Blizzard, they have performed a massive number
16 of independent genetic tests, which
17 statistically need to be corrected for that
18 because you can see associations by chance
19 alone.

20 You have cherry-picked two of
21 the things that they looked at and to be able
22 to see that that is consistent with what
23 you're seeing here and that is possible that
24 it is only by chance alone.

25 **Q. So you think --**

1 MS. BROWN: Well, please let
2 her finish. She was not finished.

3 QUESTIONS BY MR. TRACEY:

4 Q. Oh, I thought you were done,
5 I'm sorry.

6 A. I'm simply outlining that there
7 are ways to rigorously assess that, but I've
8 not seen that Carter has performed those
9 studies.

10 Q. So what I'm asking, though,
11 Doctor, is if the label is correct, okay,
12 let's assume for our purposes that the weight
13 of the evidence supports a causal association
14 between Depakote and autism and ADHD. Let's
15 pretend it's true for a second.

16 Are you with me?

17 A. I understand your assumption.

18 Q. Okay. My question is, forget
19 about all of the -- maybe they did everything
20 wrong, everything -- Carter and Blizzard did
21 everything wrong, but still, they had a very
22 high enrichment score. And if they did
23 everything wrong, that would have to be a
24 coincidence based on this label, wouldn't it?

25 MS. BROWN: Objection. Vague.

1 THE WITNESS: Given the number
2 of independent tests that they
3 performed, there is a high likelihood
4 of many things being coincidence.

5 QUESTIONS BY MR. TRACEY:

6 Q. So you think, then, the Carter
7 and Blizzard 130 enrichment score, the highest
8 of any drug that they ran through the
9 database, and the label saying that Depakote
10 causes autism, is simply a coincidence?

11 A. I'm simply saying that I don't
12 have the data to independently answer the
13 question that I've {sic} asked, and so I
14 can't assess whether it's coincidence or
15 statistically meaningful.

16 Q. Okay. Okay. Who is Brandon
17 Pearson?

18 A. Brandon Pearson is -- I believe
19 at this time he's an assistant professor at
20 the School of Public Health at Columbia.

21 Q. Did you work with Dr. Pearson?

22 A. I did.

23 Q. Do you know him to be a
24 scientist of impeccable integrity?

25 A. I wouldn't say that I know one

1 way or the other in terms of integrity.

2 Q. Really? How long did you work
3 with him?

4 A. Approximately two years.

5 Q. And in those two years of
6 working with Dr. Pearson, you were not able
7 to assess his integrity?

8 A. No.

9 Q. You published papers with
10 Dr. Pearson, right?

11 A. I've published one paper with
12 Dr. Pearson.

13 Q. You published a paper with him,
14 what was it, last month, two months ago?

15 A. I believe that's when it came
16 out.

17 Q. Dr. Pearson, in your
18 interaction with him in the two years at
19 Columbia, did he appear to be a competent
20 toxicologist?

21 A. I can't comment on his
22 toxicology experience.

23 Q. Can you comment about him at
24 all in any respect, or is he a stranger to
25 you?

1 A. I've met with Dr. Pearson, to
2 my knowledge, on two occasions.

3 **Q. Really?**

4 **How did you choose him, if you**
5 **did, to do the study?**

6 A. I did not choose him. He chose
7 me.

8 **Q. Ah, he chose you. I see.**

9 **Do -- yeah, I will.**

10 **What about Brennan Baker? Do**
11 **you know Dr. Baker?**

12 A. I believe he's a graduate
13 student in Dr. Pearson's lab or was at the
14 time.

15 **Q. Okay. Any opinion on his**
16 **scientific integrity?**

17 A. I don't really know.

18 **Q. What about your other coauthor,**
19 **Dr. Zhang?**

20 A. I don't know Dr. Zhang at all.

21 **Q. What about Jeremy Simon?**

22 A. I don't know Dr. -- or I assume
23 it's Dr. Simon.

24 **Q. Or Sarah McLarnan?**

25 A. I don't know. I don't know

1 that person personally.

2 **Q. You said you've only met with**
3 **Dr. Pearson one -- two times in your life?**

4 A. That's correct.

5 **Q. You guys were both at Columbia?**

6 A. That's correct.

7 **Q. Were you in the same**
8 **department?**

9 A. No.

10 **Q. What department were you in?**

11 A. Pediatrics.

12 **Q. Oh.**

13 A. He's in the School of Public
14 Health.

15 **Q. Have you exchanged e-mails with**
16 **Dr. Pearson?**

17 A. We have.

18 **Q. More than two?**

19 A. I don't know the number, but
20 more than two.

21 **Q. Since you left Columbia, have**
22 **you exchanged e-mails?**

23 A. No.

24 **Q. Okay. Why did you exchange**
25 **e-mails?**

1 A. About this manuscript.

2 Q. Is that the only communication
3 you've ever had with Dr. Pearson in your
4 life, was over the manuscript we're about to
5 discuss?

6 A. I won't say it's an absolute
7 that I've never, ever discussed anything with
8 him, but the vast majority of our
9 communications were around this manuscript.

10 Q. Dr. Pearson has done studies on
11 acetaminophen and neurotoxicology, hasn't he?

12 A. I -- I'm not aware of his
13 studies. Those -- I did not do any studies
14 with him with acetaminophen.

15 Q. You're not aware of his
16 research on acetaminophen?

17 A. I'm not.

18 Q. I think it's in your report,
19 isn't it?

20 A. I don't recall. Maybe you can
21 point it out to me.

22 Q. Okay. We'll get to it. We're
23 going to get to in just a second.

24 (Chung Exhibit 333 marked for
25 identification.)

1 QUESTIONS BY MR. TRACEY:

2 Q. All right. Let's -- this -- my
3 next exhibit is 333. This is a study that
4 you yourself are on, published in the journal
5 **Frontiers.**

6 A. Okay.

7 Q. This was published less than a
8 month ago, August 3, 2023.

9 And there we see Wendy Chung,
10 don't we?

11 A. Yes.

12 Q. Now, at the time you were still
13 at Columbia, weren't you?

14 A. Yes.

15 Q. And the name of the paper is,
16 "Environmental carcinogens disproportionately
17 mutate genes implicated in neurodevelopmental
18 disorders."

19 That's the name of it?

20 A. That's the title, that's
21 correct.

22 Q. And the lead author is Brennan
23 Baker, and Brandon Pearson is the senior
24 author at the end, right?

25 A. That's correct.

1 **Q. And you were there right before**
2 **Brandon, right?**

3 A. That's correct.

4 **Q. The Columbia University**
5 **actually has a mouse lab, doesn't it?**

6 A. Several investigators at
7 Columbia use rodent models, including mice.

8 **Q. Are you familiar with the lab**
9 **at Columbia that studies neurotoxicology in**
10 **mouse models?**

11 A. Not specifically. Maybe you
12 can be more specific.

13 **Q. Okay. No, I'll come back to**
14 **that. I thought you might -- you might be**
15 **familiar with it.**

16 In any event, this article was
17 published less than 30 days ago. 27 days
18 ago, right?

19 A. Let's see. It looks like
20 August 3, 2023.

21 **Q. Did you read this study before**
22 **it was published?**

23 A. Yes, I did.

24 **Q. Did you edit it?**

25 A. Yes, I did.

1 **Q. You redline it?**

2 A. Track changes, but, yes.

3 **Q. Yeah, that's track changes.**

4 **Do you believe what's written**
5 **in this paper?**

6 A. You know, there are always
7 several rounds of edits that go through any
8 manuscript. Ultimately, Dr. Pearson was the
9 one who decided the consensus and what to
10 submit in terms of the final manuscript.

11 **Q. But my question wasn't that.**
12 **It was whether you believed what's in the**
13 **paper that has your name on it that was**
14 **published less than 30 days ago with**
15 **Dr. Pearson.**

16 A. Dr. Pearson, Dr. Baker and the
17 team were the ones who performed the actual
18 experiments, so I was not involved in the
19 actual generation of the data. I was
20 involved in terms of editing the manuscript.
21 And, again, the ultimate decision about what
22 changes, what suggestions were accepted, were
23 Dr. Pearson's decisions.

24 **Q. Can you not answer my question?**

25 MS. BROWN: Objection.

1 Argumentative.

2 She did.

3 THE WITNESS: So there could
4 have been edits that I suggested as we
5 were going through multiple rounds of
6 revision. And as the senior author,
7 Dr. Pearson was the one who ultimately
8 decided how the final manuscript is as
9 you see it today.

10 QUESTIONS BY MR. TRACEY:

11 Q. All of that may be true. My
12 question to you, is the published work that,
13 after all the edits, after all the redlines
14 that was published for the world to see, is
15 it true, best you can tell?

16 A. I think the major takehome
17 message, yes, I stand behind the major
18 conclusions of the manuscript.

19 Q. Is there some part of it you
20 don't stand behind, Dr. Chung?

21 A. Again, in terms of the major
22 takehome message and to the best of my
23 knowledge in terms of the data as they were
24 presented to me, I stand behind the
25 conclusions of this manuscript.

1 **Q. When did you get hired by**
2 **Johnson & Johnson, Dr. Chung?**

3 MS. BROWN: Objection.

4 THE WITNESS: I've not been
5 hired by Johnson & Johnson.

6 QUESTIONS BY MR. TRACEY:

7 **Q. Who hired you?**

8 A. I've been retained by the legal
9 team to be able to render a report and an
10 opinion.

11 **Q. When did you get hired by the**
12 **legal team in this case?**

13 A. Approximately one year ago was
14 when I was retained.

15 **Q. Okay. And how much do you get**
16 **paid? I should have asked that. I'm sloppy.**
17 **How much do you get paid an**
18 **hour?**

19 A. \$600 an hour.

20 **Q. And how many hours do you have**
21 **in this case?**

22 A. I don't know. I've submitted
23 invoices, and we can add those up.

24 **Q. Okay. Are you doing any other**
25 **work for law firms right now on other cases?**

1 A. Yes.

2 **Q. How many?**

3 A. A total of five, at one stage
4 or another.

5 **Q. And those five cases, are they**
6 **all for drug companies?**

7 A. No, none of them are for drug
8 companies other than -- I shouldn't say drug
9 companies, but anything involving medication
10 or a product.

11 **Q. Are they medical malpractice**
12 **cases?**

13 A. Yes.

14 **Q. Where somebody is alleging that**
15 **some birth trauma or something else**
16 **contributed to cause a -- cause autism, and**
17 **you're stepping in to say it was all genetic?**

18 A. No.

19 **Q. Okay.**

20 A. But they are medical
21 malpractice cases.

22 **Q. In all of those cases, is it**
23 **your opinion that the cause of the autism was**
24 **genetic?**

25 MS. BROWN: And I would just be

1 careful if your opinion hasn't been
2 disclosed in those cases, please do
3 not provide --

4 MR. TRACEY: I'm not asking you
5 to identify the case. I don't care.

6 THE WITNESS: So those cases do
7 not involve autism.

8 QUESTIONS BY MR. TRACEY:

9 **Q. Oh, what do they involve?**

10 A. Other medical problems.

11 **Q. Like what?**

12 A. I don't know what I'm allowed
13 to disclose. I'll just say that in general.
14 But I'll just say they're, you know, problems
15 in terms of medical outcomes.

16 **Q. Well, you can disclose anything**
17 **because I haven't asked you to identify**
18 **anyone.**

19 A. Okay.

20 **Q. So there's no privacy issues.**

21 MS. BROWN: Well, but I think
22 there might be a confidentiality issue
23 if you're able to reverse-engineer
24 what cases she's been disclosed in and
25 her opinion has not yet been given.

1 MR. TRACEY: Fair enough. I --

2 MS. BROWN: So I would just be
3 careful --

4 MR. TRACEY: I got it.

5 MS. BROWN: Okay.

6 QUESTIONS BY MR. TRACEY:

7 Q. None of those cases involve
8 autism?

9 A. No.

10 Q. Okay. Okay. \$600 an hour, you
11 got hired a year ago by a law firm.

12 Who does the law firm
13 represent?

14 A. Eva Canaan. You can, I assume,
15 figure out which law firm she is.

16 MR. WATTS: King & Spalding.

17 MR. TRACEY: Oh, King &
18 Spalding. Interesting.

19 QUESTIONS BY MR. TRACEY:

20 Q. All right. Let's look at your
21 paper. And the Results section on the first
22 page say, "We demonstrate that several
23 mutagens, including radiation and polycyclic
24 aromatic hydrocarbons, disproportionally
25 mutate genes related to neurodevelopmental

1 **disorders including autism spectrum**
2 **disorders, schizophrenia,**
3 **attention-deficit/hyperactivity disorder."**

4 **Did I read that correctly?**

5 A. Yes, that's the first sentence
6 under the Results.

7 **Q. Did you and your coauthors**
8 **demonstrate that in your study?**

9 A. Within -- with this system,
10 yes.

11 **Q. Okay. So you demonstrated that**
12 **environmental factors disproportionately**
13 **mutated genes related to neurodevelopmental**
14 **disorders?**

15 A. Again, for mutagens, radiation
16 is obviously a very strong mutagen. Genes
17 that are related to the conditions that they
18 describe are relatively large genes. The
19 larger the gene is, the bigger the target it
20 is for something that alters DNA.

21 And so in that case, yes, those
22 were shown in this system to be associated
23 with an increased chance.

24 **Q. And polycyclic aromatic**
25 **hydrocarbons, that's pollution, isn't it?**

1 A. That is part of what can be
2 found in pollutants.

3 Q. Uh-huh.

4 Flip over to the introduction,
5 because you begin your introduction citing
6 two other papers -- three other papers there.

7 And the one -- I want to focus
8 on that Pugsley 2021 that you cited.

9 Do you see that?

10 A. Yes.

11 Q. Now, you cited Pugsley in this
12 paper four or five times.

13 Did you know that?

14 A. We could go back and count, but
15 that wouldn't surprise me.

16 Q. Do you know the Pugsley 2021
17 paper?

18 A. Yes, I see the paper.

19 Q. Pardon me?

20 A. Yes, I see the paper in the
21 references.

22 Q. Yeah.

23 But are you familiar with the
24 paper that you cited -- I think the record is
25 going to reflect four or five times -- in

1 **this paper?**

2 A. I do see the paper. I don't
3 recall all the details off the top of my
4 head, but I do see the paper.

5 Q. And you-all cited Landrigan in
6 this paper, too? Remember we talked about
7 Philip Landrigan? That was the first article
8 we looked at, I think.

9 A. Yes, I see --

10 Q. Do you see Landrigan?

11 A. I see the citation.

12 Q. And you cited him for the
13 proposition, you did, "Similarly,
14 environmental exposures may be responsible
15 for a large proportion of neurodevelopmental
16 disorders."

17 That's what you said, right?

18 A. Again, the team of authors had
19 this written here.

20 Q. Okay. Do you want to take that
21 back now?

22 A. I'm not taking it back, but I
23 am saying that this manuscript was driven by
24 Drs. Baker and Pearson being the first and
25 the senior authors.

1 **Q.** **Okay. That's fine.**

2 **But is it true what they wrote?**

3 **A.** I think they have referenced a
4 paper.

5 **Q.** **Okay. You see Pugsley again**
6 **there.**

7 **You cite him again next to**
8 **Kinney?**

9 **A.** Yes, I see the second one.

10 **Q.** **"Environmentally induced**
11 **mutation remains a strong yet generally**
12 **untested candidate mechanism that may link**
13 **environmental exposures to neurodevelopment."**

14 **And there's Pugsley 2021 again,**
15 **isn't it?**

16 **A.** Yes. Again, "untested," I
17 think, is a key word here.

18 **Q.** **Pardon me?**

19 **A.** I think "untested" is one of
20 the key words here.

21 **Q.** **Indeed.**

22 **You were doing the testing in**
23 **this study, weren't you?**

24 **A.** We were using a system to test,
25 yes.

1 **Q. Yes.**

2 **And the system you used to test**
3 **proved that the environment was creating de**
4 **novo mutations?**

5 A. That certain things could
6 create de novo mutations, certain exposures.

7 **Q. And you stand behind everything**
8 **in this paper, don't you, Dr. Chung?**

9 A. As I said --

10 MS. BROWN: Object to the form.

11 THE WITNESS: As I said
12 earlier, the major conclusions, yes, I
13 do stand behind.

14 (Chung Exhibit 313 marked for
15 identification.)

16 QUESTIONS BY MR. TRACEY:

17 **Q. Okay. Good for you.**

18 **Let's pull up Pugsley. This is**
19 **Exhibit 314. We're going to talk about**
20 **Pugsley and see what these authors who you**
21 **cited four times have to say about the**
22 **subject that we're here to talk about.**

23 MR. WATTS: 314?

24 MR. TRACEY: 314.

25 Pugsley. I have 314. Is that

1 wrong?

2 THE WITNESS: This looks like
3 something else.

4 MR. TRACEY: 313? My bad. I
5 gave you the wrong one. Danny's bad.
6 Mine says 314. It's 313.

7 Thank you, Senor Watts.

8 QUESTIONS BY MR. TRACEY:

9 Q. By the way, were you Brandon
10 Pearson's postdoc advisor?

11 A. No.

12 Q. No?

13 Oh, Zylka, I see.

14 Do you know Dr. Zylka?

15 A. I do know Dr. Zylka.

16 Q. Were you her postdoc advisor?

17 A. I believe it's Mark Zylka, if
18 it's the same Zylka I'm thinking of, and --

19 Q. Did I say "her"? Oh, I'm
20 sorry.

21 A. And, no, I was -- I've not
22 directly worked with Mark Zylka.

23 Q. Okay. Let's turn to -- see
24 what the good doctors, Pugsley, et al., have
25 to say.

1 By the way, this was published
2 in 2022, I think, in the fall -- October.
3 There we go. October of 2022. This was
4 published -- yeah, 20 -- yeah, it was
5 published in 2022 in Molecular Psychiatry.

6 Are you familiar with that
7 journal?

8 A. I am familiar with the journal.

9 Q. Is that a high impact journal?

10 A. We would have to look at the
11 impact score.

12 Q. Okay.

13 A. I don't believe it's greater
14 than 10, but I don't recall.

15 Q. Well, in any event, this is the
16 paper you cited four times, right?

17 A. The paper cited in the
18 manuscript, correct.

19 Q. Okay. The name of it is
20 "Environmental exposures associated with
21 elevated risk for autism spectrum disorder
22 may augment the burden of deleterious de novo
23 mutations among probands."

24 That's the name of the article,
25 right?

1 A. That's the title, correct.

2 Q. That's the title.

3 Now, I notice with some
4 interest that your report, you don't cite
5 Pugsley in your expert report in this case
6 even one time, do you?

7 A. No, I don't believe I did.

8 Q. How is it that you -- that this
9 paper escaped your search criteria when you
10 were reviewing the relevant literature?

11 A. I'm sorry, can you rephrase the
12 question?

13 Q. How is it -- whatever search
14 criteria that you used, how is it that you
15 didn't capture Pugsley as a relevant piece of
16 literature?

17 MS. BROWN: Objection to the
18 form.

19 THE WITNESS: Well, for one
20 thing, I believe this is a review
21 article.

22 QUESTIONS BY MR. TRACEY:

23 Q. Yes, ma'am, it is.

24 A. So I --

25 Q. Okay.

1 A. In general, I'm trying to find
2 the primary source data to be able to
3 directly assess the source of the data.

4 **Q. So if I look through your**
5 **report, you will only have referenced**
6 **original sources? There will be no review**
7 **articles, there will be no commentaries,**
8 **there will be no reviews?**

9 MS. BROWN: Objection.
10 Misstates testimony.

11 THE WITNESS: No, I'm not
12 saying there isn't a review, but in
13 terms of the things that one is trying
14 to gather the evidence and assess the
15 evidence, one should directly look at
16 the evidence.

17 QUESTIONS BY MR. TRACEY:

18 **Q. Well, how did one end up with**
19 **review articles in your paper and miss**
20 **Pugsley?**

21 A. In certain cases, there were
22 reviews, for instance, of genetics. I have a
23 very long report and a very long section on
24 genetics. And so many of those have
25 references to reviews, more so than, for

1 instance, looking at individual
2 paper-by-paper, the direct epidemiological
3 data.

4 Q. Well, let's see what they have
5 to say.

6 The very first sentence in the
7 abstract says, "Although the full etiology of
8 autism spectrum disorder is unknown, familial
9 and twin studies demonstrate high
10 heritability of 60 to 90 percent, indicating
11 a predominant role of genetics in the
12 development of the disorder."

13 Do you agree with that?

14 A. I agree about the predominance
15 of genetics and this developmental disorder,
16 autism.

17 Q. In fact -- in fact, that's
18 almost word for word your conclusion, isn't
19 it?

20 A. I don't know about word for
21 word, but --

22 Q. Well, let me read it for you.

23 You say, "Genetics are the
24 predominant cause of ASD and ADHD."

25 They say "predominant role."

1 A. In that sense, yes, we do both
2 use the word "predominant."

3 **Q. So the authors, and at least**
4 **Dr. Chung on that issue, seem to be in**
5 **agreement?**

6 A. I think we both have an opinion
7 that genetics are the predominant
8 contributing factor to autism.

9 **Q. But I think you may have a**
10 **disagreement on what that means, Dr. Chung.**
11 **Don't you?**

12 A. You'll have to be more
13 specific.

14 **Q. Well, you've read this, ma'am,**
15 **haven't you, lately?**

16 A. If you want to ask a specific
17 question, I'm glad to consider it.

18 **Q. I just asked a specific**
19 **question.**

20 **You read this article recently,**
21 **didn't you?**

22 A. Yes, I've read this article.

23 **Q. When's the last time you read**
24 **it?**

25 A. Within the last six months.

1 **Q. What about in the last six**
2 **days?**

3 A. I don't think I've read it
4 cover to cover in the last six days, no.

5 **Q. But you've read it in the last**
6 **six days, didn't you, Dr. Chung?**

7 MS. BROWN: Objection.

8 THE WITNESS: I don't
9 understand the question. I have not
10 read it start to finish in the last
11 six days.

12 QUESTIONS BY MR. TRACEY:

13 **Q. I didn't ask you about start to**
14 **finish, Doctor.**

15 **You have laid your eyes on this**
16 **paper in the last six days, haven't you?**

17 MS. BROWN: Objection. Asked
18 and answered.

19 THE WITNESS: I don't recall
20 looking at this in the last six days.
21 I'm not sure why the last six days is
22 coming up.

23 QUESTIONS BY MR. TRACEY:

24 **Q. Okay. Well, let's see what**
25 **they say. They say, "The genetic**

1 architecture of ASD consists of a complex
2 array of rare and common variants of all
3 classes of genetic variation usually acting
4 additively to augment individual risk."

5 And you certainly agree with
6 that, right?

7 A. I agree that it's complex, and
8 they're both rare and common genetic
9 variants, yes.

10 Q. And then they say there -- they
11 start off a sentence, "Notably."

12 Do you see that? "Notably" --
13 oops.

14 "Notably, environmental
15 exposures attributed as salient risk factors
16 for ASD may play a causal role in the
17 emergence of deleterious de novo variations
18 with several ASD-associated agents having
19 significant mutagenic potential."

20 Do you remember reading that
21 when you reviewed this paper?

22 A. Yes, and I would, again,
23 underscore the word "may" within this. I
24 think if you were to ask these authors, they
25 would say the proven association is with

1 advanced paternal age with de novo genetic
2 variance.

3 Q. I didn't hear the last part.

4 A. I think if you ask these
5 authors, they would say that the one
6 established association is with advanced
7 paternal age with de novo genetic variance.

8 Q. Okay. Well, let's not guess
9 because they're not here. Let's see what
10 they wrote in the paper.

11 Okay?

12 A. Okay.

13 Q. Salient means important,
14 doesn't it?

15 A. I would say relevant.

16 Q. Okay. So environmental
17 exposures attributed as important risk
18 factors may play a causal role in the
19 emergence of deleterious de novo variations,
20 right?

21 A. That's what they say.

22 Q. And then they say, "Broadly,
23 these exposures were observed to elicit
24 genomic alterations through one or a
25 combination of, 1, direct interaction with

1 **genetic material."**

2 **Would that be -- would that be**
3 **a mutagen?**

4 A. I think they are hypothesizing
5 that mutagens that alter DNA could have this
6 effect.

7 Q. And then number 2, they have,
8 **"Impaired DNA repair."**

9 **What do we call that?**

10 A. Impaired DNA repair.

11 Q. Okay. Not as complex as I
12 **thought.**

13 **And 3 is, "Oxidative DNA**
14 **damage."**

15 **Right?**

16 A. Correct.

17 Q. By the way, do you know what
18 the reputation of Monash University in
19 Australia is in the worldwide research
20 community?

21 A. No, I don't.

22 Q. Okay. So down at the bottom of
23 the first paragraph to the right, they start
24 talking about the relative contributions of
25 mutations to the etiology of autism.

1 Do you see where they say, "The
2 relative contribution of these mutations to
3 the etiology of the disorder is estimated at
4 2.5 to 15 percent and 12 to 52 percent
5 respectively, with more recent evidence
6 supporting the role of tandem repeat
7 variations as additional and incredible
8 salient components of the ASD genotype"?

9 A. I see the sentence, yes.

10 Q. Do you agree with their -- what
11 they're writing about what the data shows?

12 A. Yes, I support -- or I believe
13 that these genetic contributions, that's
14 approximately the right percentages.

15 Q. Yeah, and they're referring us
16 to the section on elevated genomic
17 sensitivity to exposure-induced mutagenesis,
18 aren't they?

19 Which we'll look at in a
20 minute.

21 A. Yes, they do reference this
22 other section.

23 Q. But up at the top of this
24 second page, I want you to look at the
25 diagram they put together for everybody.

1 And it's called "ASD etiology."
2 They've spelled it "aetiology"
3 because they're Australian, but etiology
4 means cause of, right?

5 A. Yes.

6 Q. So we're looking at ASD, the
7 cause, according to these researchers that
8 you cited in your most recent paper.

9 And under Figure 1, they tell
10 us what we're looking at. It's a
11 diagrammatic representation of the interplay
12 between genetic and environmental risk
13 factors in the etiology of ASD. That's all
14 bolded there.

15 Do you see that?

16 A. Yes, I see that.

17 Q. And then they say, "Both
18 heritability and nonheritable factors can
19 independently and reciprocally influence the
20 development of ASD symptomatology."

21 I'm going to stop there for a
22 second. Do you agree with their statements
23 there?

24 A. I agree that both heritable and
25 nonheritable factors can influence the

1 development of ASD.

2 **Q. Reciprocally?**

3 A. I'm not sure I understand
4 exactly without reading in detail, which we
5 can stop and I can do, but I need to look at
6 further what they mean by reciprocally.

7 **Q. Okay. With that understanding,**
8 **let's move on. "Up to 5 to 15 of ASD**
9 **probands possess risk-associated de novo**
10 **mutations, indicating the significance of**
11 **nonfamilial genetic variability in**
12 **disordering -- in determining disorder risk."**

13 **And you certainly agree with**
14 **that?**

15 A. Yes.

16 **Q. And then finally on this they**
17 **say, "The mutagenic genotoxic potential of**
18 **nonheritable factors associated with ASD**
19 **suggests that these toxicants may play a role**
20 **in the elicitation of spontaneous mutations."**

21 **Do you agree with that?**

22 A. I agree that they're saying --
23 and again, I'll underscore the word,
24 "suggests" and "may."

25 **Q. Yes.**

1 A. So I think they have hypotheses
2 that they would like to see tested.

3 Q. I'm going to hold you to
4 "suggest" and "may," Dr. Chung, for later in
5 the day when we look at some literature you
6 relied on.

7 Would that be okay?

8 MS. BROWN: Objection to the
9 form.

10 THE WITNESS: We can look at
11 this -- other topics this afternoon,
12 sure.

13 QUESTIONS BY MR. TRACEY:

14 Q. Okay. In the middle of this
15 first paragraph there, starting in the middle
16 it says -- sorry.

17 At the beginning it says, "The
18 contribution of hereditary in ASD persists
19 despite strong selective pressures against
20 the deleterious genetic events associated
21 with disorder onset.

22 "Interestingly, the
23 reproductive challenges faced by individuals
24 with ASD are not physical but instead mostly
25 social. Fecundity is consequently reduced

1 **among affected individuals."**

2 **What's fecundity mean?**

3 A. Reproductive, reproducing.

4 **Q. Yeah. What they're saying is**
5 **even though people that have autism spectrum**
6 **disorder don't reproduce, yet it's**
7 **persisting, right?**

8 A. For some individuals.

9 **Q. Yeah.**

10 **They say, "Despite this, the**
11 **prevalence of ASD is demonstrated stability**
12 **or increases over time with nonheritable**
13 **etiological factors insufficiently**
14 **compensating for the loss of high-risk**
15 **genetic variants from the reproductive gene**
16 **pool."**

17 **Do you know what they're saying**
18 **there?**

19 A. Yes. They're saying that
20 individuals -- often those individuals with
21 intellectual disabilities and autism, are not
22 having children.

23 And to understand how those
24 individuals continue in a population genetic
25 point of view, there are de novo mutations

1 that largely account for those individuals.

2 Those de novo mutations
3 oftentimes, as I was saying before, are
4 associated with advanced paternal age.

5 And as we've seen, the
6 reproductive habits of humans change over
7 time, and as we've seen individuals
8 reproducing older, people have hypothesized
9 that that's where portions of the autism
10 population are coming from.

11 **Q. But of course the name of this**
12 **paper is not paternal age; it's environmental**
13 **exposures, isn't it?**

14 **A.** That is the title of this
15 paper, but I'm also explaining the very
16 well-established association of de novo
17 mutations with advanced parent age.

18 **Q. I have no quibble with you**
19 **there, Dr. Chung. None.**

20 They go on to say,
21 "Preservation of the genetic liability of ASD
22 despite reduced transmission of risk variants
23 has been theorized to occur, in part, due to
24 spontaneous de novo mutations," and then they
25 cite us to Table 1, right?

1 A. Yes.

2 Q. And then if we flip over to the
3 next column -- not flip, just look over to
4 the paragraph at the top that starts off
5 "environmental exposures."

6 Do you see that?

7 A. Yes, I see that.

8 Q. They say, "Environmental
9 exposures that are classically attributed as
10 salient risk factors."

11 There's that "salient" again,
12 isn't it?

13 A. Yes.

14 Q. "For ASD and other
15 neurodevelopmental disorders could represent
16 a catalyst for deleterious de novo variation
17 with several disorder-associated agents
18 having significant mutagenic and genotoxic
19 potential.

20 "However, although the
21 neurotoxicity and teratogenicity conferred by
22 these toxicants is well-established, their
23 potential role in the genesis of the de novo
24 mutations of relevance to ASD has received
25 little attention."

1 And then this is the part I
2 want you to concentrate on.

3 "Toxicogenomic analyses suggest
4 disorder-associated exposures may perturb ASD
5 susceptibility genes through mutagenic
6 chemical-gene interactions."

7 Do you see that, ma'am?

8 A. I see that statement.

9 Q. Who do you think Pugsley is
10 citing for that proposition? I'll give you
11 two guesses.

12 A. I don't know that we have to
13 guess. We just have to look at the
14 reference.

15 Q. The first name rhymes with
16 martyr.

17 A. Carter.

18 Q. Carter and Blizzard, right?

19 A. Yes, that's the reference.

20 Q. In 2022, Pugsley, the group you
21 cited, cited to this very paper -- cite
22 Carter and Blizzard for that proposition,
23 don't they?

24 A. Notably. What they don't cite,
25 though, are data -- epidemiological data

1 demonstrating an association in people of
2 known mutagens or carcinogens and associated
3 with an increased risk of autism.

4 Q. Well, how do you know they
5 don't do that?

6 I thought you hadn't read the
7 paper recently.

8 A. I'm pointing out that the
9 reference that you alluded to here was, in
10 fact, the Carter paper, was not a paper in
11 terms of looking at the epidemiological
12 studies.

13 Q. Okay. Let me ask you this. Do
14 you disagree with Pugsley when they say,
15 "Toxicogenomic analysis suggests
16 disorder-associated exposure may perturb
17 known ASD susceptibility genes through
18 mutagenic chemical-gene interactions"?

19 MS. BROWN: Well, I object to
20 the partial reading of that sentence.

21 THE WITNESS: Again, what
22 they're -- what they have stated is
23 the toxicogenomic analyses, which are
24 the Carter analyses that were
25 performed. What they are not citing

1 and they, in fact, say that there's a
2 paucity of evidence in terms of being
3 able to look at this in people and
4 with epidemiological data.

5 If this were a strong factor, I
6 would have expected that someone would
7 have observed clinically this
8 observation already and that it would
9 have been studied in epidemiological
10 studies.

11 QUESTIONS BY MR. TRACEY:

12 Q. How many epidemiology studies
13 have looked at acetaminophen exposure and
14 neurodevelopmental disorders?

15 A. I can't count the number, but
16 there are several.

17 Q. More than 20?

18 A. I believe so.

19 Q. All right. Let's soldier on.

20 So on page 714 of Pugsley,
21 sorry, he referred us to Table 2 in that --
22 that sentence, which is why I'm -- let's
23 finish that sentence. I should have read the
24 whole thing. You guys are right. Let me
25 read it. Go back. Sorry, Mike, to where we

1 **were.**

2 **So, "Toxicogenomic analyses**
3 **suggest disorder-associated exposures may**
4 **perturb known ASD susceptibility genes**
5 **through mutagenic chemical-gene**
6 **interactions."**

7 **And that's Carter and Blizard.**

8 **"However, a paucity of evidence**
9 **limits the current conceptualization of this**
10 **relationship to a very few exposures," and**
11 **then they cite us to Table 2 for those**
12 **exposures.**

13 **Okay? And so now I want to**
14 **look at Table 2 where they told us to go**
15 **look.**

16 **Okay? Are you with me?**

17 A. I'm on Table 2.

18 Q. **And it's called "Environmental**
19 **exposures associated with elevated ASD risk**
20 **and evidence of their potential mode of**
21 **mutagenicity/genotoxicity in vivo and/or**
22 **in vitro."**

23 **That's what they call this**
24 **table?**

25 A. That's the title of the table.

1 Q. And of course at the top, we
2 see pollution, which we talked about, right,
3 air pollutants? Right?

4 A. Yes, there are air pollutions
5 under the category Air Pollutants.

6 Q. And then if we want to look at
7 parental health factors, which is down there,
8 we see that.

9 You want to talk about that,
10 and that's there, isn't it?

11 A. Yes, increasing parental age is
12 there.

13 Q. Yeah.

14 And they say, "Increasing
15 parental age at the time of conception is
16 associated with an increased risk of ASD.
17 Odds ratio 1.41, maternal; paternal, 1.55,"
18 right?

19 A. Correct.

20 Q. Those are modest in your world,
21 right?

22 A. They are.

23 Q. Okay. And then, of course, we
24 see heavy metals, and then we see "Inference
25 of endogenous DNA repair responses," and you

1 **see they've got oxidative DNA damage?**

2 A. Yes, they have oxidative DNA
3 damage.

4 Q. And then they list the
5 pollutants under the oxidative DNA damage,
6 don't they?

7 A. Correct.

8 Q. All right. And then if we go
9 to page 716, the table goes on for a few
10 pages, and we have pre and postconceptual
11 drug use.

12 And there, again, on another
13 table, with other researchers, we see
14 acetaminophen, don't we?

15 A. Acetaminophen is listed.

16 Q. And right underneath it is
17 thalidomide, right? Side by side or
18 underneath.

19 A. Yes, I do see thalidomide
20 underneath that.

21 Q. And under the link to ASD, they
22 say, "Meta-analytical review indicates
23 maternal use of paracetamol during pregnancy
24 increases risk of ASD in offspring, relative
25 risk 1.19. And elevated biomarkers of

1 **paracetamol metabolites in umbilical cord**
2 **plasma is associated with childhood ASD, an**
3 **odds ratio of 2.14."**

4 **Do you see that, ma'am?**

5 A. I do see that. I would say the
6 exposure, in particular with the umbilical
7 cord plasma samples, I have trouble in terms
8 of making a story that puts together that
9 timing of exposure and the risk that we're
10 talking about, given that the hypothesis here
11 is that it's invoking DNA damage.

12 Q. Okay. Well, I might be able to
13 help you with that concept when we get to Ji.

14 But in any event, Pugsley
15 references those studies and says that
16 paracetamol has been -- the link to ASD for
17 paracetamol is the meta-analysis and then Ji,
18 right?

19 A. I'm sorry, can you ask that
20 again?

21 Q. Yeah.

22 Pugsley cites two studies in
23 human beings that they say links
24 acetaminophen to autism.

25 A. Correct. References 29 and

1 115, is that what you mean?

2 Q. Yes, ma'am.

3 A. Yes.

4 Q. And then to the right under
5 "Evidence of mutagenicity, genotoxicity,"
6 they say, "Oral doses of therapeutic doses of
7 paracetamol induces oxidative stress-related
8 gene expression in peripheral blood in humans
9 in vivo; and elevated concentrations of
10 urinary" -- "urinary paracetamol is
11 associated with increased DNA fragmentation
12 in human sperm in vivo."

13 A. That is what they state, but
14 what I would say is notably absent is what
15 they don't state. Given that acetaminophen
16 is used quite widely, there are ample
17 opportunities to directly demonstrate
18 evidence of accumulated DNA mutations in any
19 number of studies that have been performed,
20 and I don't see any evidence of that effect.

21 Q. Has anybody looked for it?

22 A. I don't know all the studies
23 that have been done.

24 Q. Have you seen even one where
25 anybody even attempted to look for it?

1 A. Again, I don't know all of the
2 studies that have been done, but notably the
3 fact that it hasn't been reported, I think,
4 is notable.

5 **Q. Do you think Johnson & Johnson**
6 **should look for that?**

7 MS. BROWN: Objection to the
8 form. Lacks foundation.

9 THE WITNESS: I'm not going to
10 make a comment about what Johnson &
11 Johnson shouldn't do.

12 QUESTIONS BY MR. TRACEY:

13 **Q. Well, who should be looking for**
14 **it?**

15 MS. BROWN: Objection to the
16 form.

17 THE WITNESS: There are many
18 large epidemiological studies that
19 have the ability to look for this
20 association; and in particular, this
21 type of variation is often seen with
22 other conditions or would be seen with
23 cancer.

24 And I've not seen, given the
25 widespread use of acetaminophen, of an

1 association, for instance, with cancer
2 associated with exposures.

3 QUESTIONS BY MR. TRACEY:

4 Q. Okay. Have you seen a study
5 that looked for and said it's not there?

6 A. I haven't done a comprehensive
7 review to look for it.

8 Q. Okay. Don't you think you
9 should do that?

10 MS. BROWN: Objection.

11 Argumentative.

12 THE WITNESS: Again, I think
13 what's notable is given the widespread
14 use and given the number of conditions
15 that could be associated, that it
16 hasn't come up and it would have been,
17 I think, very visible to me and to the
18 general public.

19 QUESTIONS BY MR. TRACEY:

20 Q. How long did it take to figure
21 out that -- how long did it take to figure
22 out that lead was poisoning children?

23 MS. BROWN: Objection to the
24 form.

25 THE WITNESS: I did not do a

1 literature review to get the history
2 of lead exposures.

3 MS. BROWN: Mr. Tracey, can we
4 take a lunch break when you get to a
5 good stopping point?

6 MR. TRACEY: Yeah, let me just
7 finish this paper.

8 QUESTIONS BY MR. TRACEY:

9 Q. Just below acetaminophen again
10 we see thalidomide, right?

11 A. Yes, I see thalidomide.

12 Q. On another list with
13 paracetamol by another group of researchers,
14 right?

15 A. There's a different reference,
16 correct.

17 Q. Yeah.

18 And of course we see mercury
19 down there. Mercury is a well-known
20 neurotoxicant, isn't it, Doctor?

21 A. I haven't explored mercury.

22 Q. You don't know that?

23 A. I haven't explored mercury.

24 Q. Okay. If we flip over on the
25 next page, we'll see pre and postconceptual

1 **drug use. We see, there we go, marijuana**
2 **again, right?**

3 A. I do see, yeah, TCH {sic}.

4 Q. **Yeah. Opioids, right?**

5 A. I see opioids listed.

6 Q. **And there's valproate again.**
7 **That's the generic name for Depakote, right?**

8 A. Valproate is listed.

9 Q. **Yeah.**

10 **And there is the -- looks like**
11 **the same studies you and I looked at in the**
12 **label, the 2.9 relative risk there for ASD.**

13 **Do you see that?**

14 A. I see that.

15 Importantly, again, what this
16 paper points out is that this is
17 hypothesis-generating with some very simple
18 experiments to test the hypotheses given our
19 ability to do DNA sequencing and directly see
20 mutations as they accumulate in individuals
21 over time.

22 Q. **Yeah, but let's be clear. The**
23 **hypothesis is whether it's a mutagen causing**
24 **de novo mutations.**

25 **They're not hypothesizing**

1 **whether there's an ASD link to acetaminophen?**

2 MS. BROWN: Objection to the
3 form.

4 THE WITNESS: And what I'm
5 saying is that exact idea about
6 whether or not they're inducing DNA
7 mutations is very easy to test, given
8 the common population exposure to
9 acetaminophen and the number of
10 genomic studies that have been
11 performed.

12 QUESTIONS BY MR. TRACEY:

13 **Q. Why doesn't Pugsley say that in**
14 **his paper? Why don't they say, "Wow, this is**
15 **real easy to figure out. Surely somebody's**
16 **figured it out. I've written a 20-page paper**
17 **about it, and I can't figure it out"?**

18 **Why isn't that in their paper?**

19 MS. BROWN: Object to the form
20 of the question. Compound.

21 THE WITNESS: I can't guess
22 what Pugsley is thinking.

23 QUESTIONS BY MR. TRACEY:

24 **Q. Well, can you think of a**
25 **scientific reason why Pugsley -- if it's so**

1 **obvious to you, why Pugsley wouldn't put that**
2 **in his paper?**

3 MS. BROWN: Objection to the
4 form. Speculation.

5 THE WITNESS: Again, I can't
6 speak for these authors in terms of
7 what they may even be doing or have
8 done in the past. I don't know.

9 QUESTIONS BY MR. TRACEY:

10 **Q. Have you counseled the lawyers**
11 **that represent Johnson & Johnson or Kenvue**
12 **that it'd be a good idea to look for that**
13 **kind of literature to find out whether their**
14 **drug that's been on the market since 1955 was**
15 **contributing to de novo mutations in humans?**

16 MS. BROWN: Objection to the
17 form.

18 THE WITNESS: Again, this is a
19 very common exposure that's included
20 within many epidemiological studies
21 completely unrelated to autism or
22 ADHD, and I've not seen any published
23 reports of an association of any
24 conditions, notably things like
25 cancer.

1 QUESTIONS BY MR. TRACEY:

2 Q. Yeah, but my question wasn't
3 about what you saw in the literature. My
4 question is about counseling the lawyers that
5 represent the defendants in this case.

6 Have you had that conversation?

7 A. I have not had that -- the
8 scope of my work has been to assess the
9 association between genetic factors,
10 acetaminophen, prenatal exposures and autism.
11 That's the scope of what I was asked to
12 render an opinion upon.

13 MR. TRACEY: Okay. Let's take
14 a break. That's a long paper. I'm
15 not done, so let's go ahead and take a
16 lunch break.

17 MS. BROWN: Sounds good.

18 VIDEOGRAPHER: The time is
19 12:40 p.m., and we're off the record.

20 (Off the record at 12:40 p.m.)

21 VIDEOGRAPHER: The time is
22 1:18 p.m., and we're on the record.

23 QUESTIONS BY MR. TRACEY:

24 Q. Hi, Doctor, are you ready?

25 A. I am.

1 Q. We were -- oh, sorry,
2 apparently I'm not.

3 We were talking about the
4 Pugsley paper before lunch, and I just had a
5 few more questions about it. It's -- I guess
6 I should identify it again for the record.
7 Exhibit 313, right?

8 A. Yes.

9 Q. 313.

10 And if we can bring it up
11 again, to Figure 2, which is page 718. The
12 next page that -- yeah, there we go.

13 The authors have another
14 diagram for us to look at, and they call it
15 "The diagrammatic representation of the
16 impact of environmental factors on genomes
17 within parental germlines and offspring."

18 And they say, "ASD-associated
19 toxicants, e.g.," -- for example --
20 "herbicides and heavy metals, can induce de
21 novo mutations in parental germline cells
22 which may be transmitted to offspring in the
23 subsequent generation."

24 And then they say, "For
25 example, induce double-stranded breaks and

1 **impaired BRCA1-directed homologous**
2 **recombination, HR, DNA damage response, DDR,**
3 **can elicit de novo mutations and hamper their**
4 **repair. Offspring may also acquire**
5 **agent-induced mutations at later stages of**
6 **development, resulting in somatic mosaicism.**
7 **Genes impacted by these processes can lead to**
8 **aberrant neural development and functioning**
9 **contributing to the onset of ASD."**

10 **What they -- what they say**
11 **there, is that accurate scientifically?**

12 MS. BROWN: Objection to the
13 form. Compound.

14 THE WITNESS: So I think they
15 have what I would describe as a life
16 course schematic with a hypothesis
17 about how DNA damage can lead to de
18 novo mutations in the offspring. So
19 I'm -- you know, I think that's what
20 they're trying to illustrate in the
21 figure.

22 QUESTIONS BY MR. TRACEY:

23 **Q. Okay. And then when we look at**
24 **the figure, at the top of the diagram, it**
25 **says, "Mutagenic ASD-associated environmental**

1 **exposures."**

2 **And then they've just,**
3 **generally speaking, listed the categories**
4 **that we've already looked at on the table.**

5 **Remember those?**

6 A. Those are the same categories
7 they had in the table.

8 Q. Yeah, air pollutants, heavy
9 metals, herbicides, industrial chemicals,
10 parental health factors and pre and
11 postconceptual drug exposure, and we looked
12 at many of those together, didn't we?

13 A. We did review those before the
14 break.

15 Q. Yes.

16 **And then the next sort of arrow**
17 **down or step in the process, they have three**
18 **potential -- is it mechanism? Is that the**
19 **right word to use when we're talking about**
20 **direct interaction or an impaired DNA damage**
21 **response or oxidative DNA damage? Are those**
22 **mechanisms through which de novo mutations**
23 **can take place?**

24 A. So I think there are different
25 ways that DNA damage can occur, and very

1 specifically there are signatures of DNA
2 damage that occur via different mechanisms,
3 and I think that's what they're trying to
4 illustrate.

5 And one can, in fact, see
6 signatures in terms of cause and effect by
7 looking at the DNA readout and seeing what
8 type -- not just that there are mutations,
9 but actually seeing the types of mutations
10 one has.

11 Q. Okay. But you're being sort of
12 granular. I'm asking as a threshold matter,
13 no matter how it happens, are the three
14 types -- descriptions there, direct
15 intervention, impaired DDR or oxidative DNA
16 damage, do we refer to those as mechanisms or
17 processes? What would we call those?

18 A. I would call those more
19 processes by which the DNA damage occurs and
20 specific types of DNA damage that occur.

21 Q. Okay. So oxidative, I know
22 from my cancer cases, which you referenced
23 cancer earlier, a biomarker of oxidative DNA
24 damage is 8-OHdG.

25 Are you familiar with that?

1 A. No.

2 Q. Okay. Do you do -- do you look
3 at cancer cases?

4 A. Clinically I do germline cancer
5 genetics, yes.

6 Q. Okay. Do you know what
7 biomarkers exist that are used to determine
8 oxidative DNA damage?

9 MS. BROWN: Objection to the
10 form.

11 THE WITNESS: Those are
12 something, I think, that are more
13 common with somatic. I think you're
14 referring to somatic cancer changes,
15 and that's not predominantly where I
16 spend my time.

17 QUESTIONS BY MR. TRACEY:

18 Q. Okay. Fair enough.

19 Okay. So we have these three
20 processes that we talked about in the middle,
21 all leading to de novo mutations according to
22 this chart, right?

23 A. Yes.

24 Q. And then if we follow it down,
25 they've got, you know, three different

1 **periods. They actually call it genotypic**
2 **consequences in developing offspring and**
3 **children, and then we have preconceptual,**
4 **that's postfertilization, right?**

5 A. No, I think pre --

6 Q. Oh, preconceptual. Sorry.

7 A. -- conception is actually prior
8 to fertilization.

9 Q. Yeah, sorry.

10 That would be like the exposure
11 **in the mother or father before conception?**

12 A. Correct. So that would affect
13 gametes.

14 Q. Yes.

15 Things like damage to the
16 **father's sperm?**

17 A. Correct, on the paternal side,
18 that's what it would be.

19 Q. Are you familiar with the Smarr
20 **study with respect to acetaminophen**
21 **damaging -- causing DNA damage in the male**
22 **sperm?**

23 MS. BROWN: Objection to the
24 form.

25 THE WITNESS: I don't believe

1 that particular study, no.

2 QUESTIONS BY MR. TRACEY:

3 Q. Okay. And then we have
4 gestational, which is of course, you know,
5 the in utero, postfertilization exposure,
6 right?

7 A. Yes, that would be in utero.

8 Q. And then of course when we
9 reach the what my OB/GYN friends call the
10 extra uterine environment, after you're
11 delivered, we have early development. These
12 are all three places where these de novo
13 mutations can occur. Three time frames,
14 sorry.

15 A. Sure.

16 Q. Okay.

17 A. And, in fact, they occur
18 through the life course as well.

19 Q. Yes. Yes.

20 All right. Now, if you flip
21 over to the references, reference 163 that
22 Pugsley cites to is a paper by Hill and
23 Cabrera.

24 163? Do you see it there?

25 A. Yes.

1 Q. Now, you know Dr. Cabrera. You
2 know who he is. He's an expert in this case?

3 A. Yes, I'm familiar with
4 Dr. Cabrera.

5 Q. And are you familiar with
6 Dr. Finnell from the Finnell-Cabrera lab in
7 Houston?

8 A. I don't personally know
9 Finnell.

10 Q. Are you familiar with him? Do
11 you know his work? Are you aware of him?

12 A. Not intimately familiar.

13 Q. Have you ever met him in a
14 meeting or talked to him?

15 A. No.

16 Q. Okay. Do you have an
17 understanding of whether the Finnell-Cabrera
18 lab has a reputation in the scientific
19 community?

20 A. I'm sure they have a
21 reputation. I'm not -- I'm not particularly
22 familiar with it.

23 Q. Okay. You're not familiar with
24 their lab and historically what they've
25 accomplished?

1 A. No.

2 Q. Okay. I'm assuming you've
3 never written a paper with Rick Finnell?

4 A. Not to my knowledge.

5 Q. Okay. The paper that Pugsley
6 cites to written by Robert and Rick and
7 others is, "Autism-like behavior and
8 epigenetic changes associated with autism as
9 consequences of in utero exposure to
10 environmental pollutants in a mouse model."

11 I'm assuming you're not
12 familiar with that study?

13 A. I don't believe I've read it,
14 no.

15 (Chung Exhibit 309 marked for
16 identification.)

17 QUESTIONS BY MR. TRACEY:

18 Q. Okay. Let's -- 309, please.
19 I'm going to hand you what's
20 Exhibit 329 -- sorry, 309.

21 MS. BROWN: We're not going
22 sequentially?

23 MR. TRACEY: Pardon me?

24 MS. BROWN: These numbers --

25 MR. TRACEY: No, of course not.

1 MS. BROWN: Okay.

2 MR. TRACEY: That would be too
3 easy and too normal. But you'll be
4 happy to know, that results in much
5 fewer -- many fewer exhibits so --

6 MS. BROWN: I'll take it.

7 MR. TRACEY: Yeah, I knew you
8 would.

9 MS. BROWN: Thank you.

10 QUESTIONS BY MR. TRACEY:

11 Q. So, Dr. Chung, this is a
12 PowerPoint from this year by Heather Volk and
13 others called, "Exploring Gene-Environment
14 Interactions in Autism Spectrum Disorder."

15 And if you turn the page and
16 look at the collaborators for this, you'll
17 see Johns Hopkins, Penn State, Kaiser
18 Permanente, University of Miami, et cetera,
19 et cetera.

20 Do you see any recognize?

21 A. Sure, there's several.

22 Q. Who?

23 A. Dani Fallin, Rebecca Landa, Joe
24 Piven.

25 Q. Joe Piven at UNC? I've

1 **forgotten.**

2 A. Yes.

3 Q. **Okay. Yeah, there he is. All**
4 **right.**

5 A. Bob Schultz.

6 Q. **Okay.**

7 A. Those are the people I know.

8 Q. **Okay. And as I said, I'll sort**
9 **of set this again, the title of this**
10 **presentation is "Exploring Gene-Environment;**
11 **Interactions in Autism Spectrum Disorder."**

12 MS. BROWN: And, Counsel, just
13 for the record, was this from a
14 website, or how did you get it?

15 MR. TRACEY: Man, I don't --
16 oh, that's a great -- that's a great
17 answer. I'm glad you asked that
18 question.

19 MS. BROWN: All right.

20 QUESTIONS BY MR. TRACEY:

21 Q. **What is SPARK?**

22 A. SPARK is Simons Foundation
23 Powering Autism Research for Knowledge.

24 Q. **Do you have anything to do with**
25 **SPARK?**

1 A. I'm the principal investigator.

2 Q. **This is from the SPARK website.**

3 A. Okay.

4 Q. **So this is off the website that**
5 **you're a principal investigator on, Doctor.**
6 **That's why I pulled it, actually. Glad you**
7 **reminded me.**

8 Joe Piven, how do you know who
9 **Joe Piven is, or how do you know Joe -- or**
10 **Dr. Piven, I should call him?**

11 A. He's a researcher in the autism
12 field.

13 Q. **Okay. Were you aware that the**
14 **website that you're a principal investigator**
15 **for presented with all of these different**
16 **collaborators this exploring gene-environment**
17 **interactions in autism spectrum disorder?**

18 MS. BROWN: I'll just object as
19 lacking foundation.

20 THE WITNESS: So to clarify,
21 this is not my study from SPARK. We
22 have invited guests that we invite to
23 be able to discuss scientific topics,
24 and we post those topics for the
25 public to be able to see, and this is

1 one of those topics. This is one of
2 these talks.

3 QUESTIONS BY MR. TRACEY:

4 **Q. I was just asking you, are**
5 **you -- were you aware of this, that it was on**
6 **your website?**

7 A. I don't recall listening to
8 this particular talk.

9 **Q. Okay. I really just have a few**
10 **questions about it. If we flip over to my**
11 **page 13, 13 of it, you'll see that Heather**
12 **Volk and the other collaborators --**

13 MS. BROWN: Sorry. Ours is not
14 paginated.

15 MR. TRACEY: Well, you're not
16 going -- these aren't complicated.
17 Trust me on this one.

18 MS. BROWN: It's right after
19 this one. Here, take mine.

20 QUESTIONS BY MR. TRACEY:

21 **Q. You see on my page 13 --**

22 A. I got it.

23 **Q. They use the Figure 1 from**
24 **Grandjean and Landrigan that you and I looked**
25 **at early in the deposition.**

1 A. Sure, I see it.

2 Q. And then on my 49, which I
3 apologize, yours isn't marked, there's a
4 slide called "Genes and environment in
5 SPARK."

6 Are you aware of the genes and
7 environment SPARK research that's going on?
8 GEARS? I think it's called GEARS?

9 A. I'll explain it in just a
10 second. I just want to make sure I've got
11 the page.

12 MS. BROWN: How much farther do
13 we go in?

14 MR. TRACEY: I don't know.
15 It's my page 49. I thought everybody
16 had paginated.

17 MS. BROWN: We'll get there.

18 THE WITNESS: On this one.
19 Okay.

20 MR. TRACEY: I'd say it's
21 three-quarters of the way through.
22 It's long.

23 THE WITNESS: Yep.

24 MR. TRACEY: Yeah, it's on the
25 screen. I mean, I --

1 THE WITNESS: So -- yeah. So,
2 let me explain how SPARK works.

3 So we have sites throughout the
4 country, clinical-affiliated sites.

5 Within this, we open this up to
6 something called research match, where
7 we try and make it easier for
8 researchers to do research with the
9 SPARK community.

10 Through that process, these are
11 not SPARK-endorsed, but we try to
12 enable, as I said, people to do
13 research more effectually and
14 efficiently, and the GEARS study is
15 one of the groups that has used our
16 research match mechanism.

17 And we send invitations to our
18 research community about participation
19 within research-matched studies such
20 as GEARS.

21 QUESTIONS BY MR. TRACEY:

22 Q. And this is -- so I found this
23 PowerPoint on the SPARK website, and you --
24 are you familiar with the Genes and
25 Environment Autism Research Study that's

1 **ongoing now?**

2 A. So this is not -- even though
3 we have it on the SPARK website, this is not
4 my study. These are the investigators, and
5 you'll note that I'm not listed as one of
6 those investigators.

7 Q. I actually don't think that's
8 true.

9 Can you turn to page 58,
10 please? It's called the GEARS network.

11 A. So the GEARS network, which is
12 different.

13 Q. Oh, I see. Okay. Fair enough.
14 You're not one of the
15 investigators though?

16 A. I'm not one of the GEARS
17 investigators.

18 Q. But if we look at the GEARS
19 network, you're on there, aren't you? You're
20 on there twice as being one of the GEARS --
21 two of the GEARS?

22 A. So maybe you can show me what
23 you're pointing to.

24 Q. Well, I'm assuming SPARK Chung
25 is you?

1 A. SPARK Chung is me, representing
2 the SPARK study.

3 **Q. Okay.**

4 A. And then I guess you're
5 pointing to SSC, Simons Simplex Collection.
6 Is that what you mean?

7 **Q. Yes.**

8 A. So in both of these, these are
9 cohorts for autism. SCC stands for Simons
10 Simplex Collection, and at the time that I
11 was at the Simons Foundation as the director
12 of clinical research, I was the
13 representative for both of those studies.

14 I'm no longer representing the
15 Simons Simplex Collection.

16 **Q. So -- but in March of this year**
17 **you were?**

18 A. As of March 2023, I was.

19 **Q. And what this PowerPoint says**
20 **the GEARS network is, it says "it's an**
21 **infrastructure for GxE."**

22 **That's genes and environment,**
23 **right?**

24 A. GxE stands for genes by
25 environment.

1 Q. And the GEARS network we see in
2 the top, "Combining advances in genomics and
3 environmental science to accelerate
4 actionable research and practice in ASD."

5 A. That is the acronym for GEAR,
6 yes.

7 Q. Okay. And it says it's got
8 18 network sites and plans to add more.

9 That's part of the
10 infrastructure, right?

11 A. That's up to those
12 investigators, but I presume those are their
13 plans.

14 Q. And then it says, "175,000
15 individuals."

16 That's a large number, isn't
17 it?

18 A. It is a large number.

19 Q. "Translational laboratory
20 models." That's part of their
21 infrastructure, right?

22 A. Again, this is their research,
23 but...

24 Q. Okay. And "mini-brain models
25 and community advisory board and outreach."

1 **That's what their**
2 **infrastructure they say consists of?**

3 A. Again, this is -- this is their
4 research, so I presume if that's what they
5 said and these are their slides, then that's
6 what they intend to do.

7 Q. And you told me earlier in the
8 deposition you've never heard of Heather
9 Volk; is that right?

10 A. I don't know her personally,
11 no.

12 Q. That's different than never
13 having heard of her.

14 My question earlier was, have
15 you ever heard of her before?

16 A. No. I mean, her name doesn't
17 ring bells for me.

18 Q. Okay. And yet here you are,
19 not once, but twice, in the GEARS network
20 where she's the principal investigator?

21 A. Again, in doing this, you can
22 see many of these cogs here, given that we
23 give many, many people access to SPARK
24 through the research match mechanism, there
25 are many people that I don't know who are

1 using our resources, hundreds of
2 investigators around the world.

3 Q. Wait. It's not just your
4 resources. There's 18 different databases
5 that are being accessed here, right?

6 A. My point being -- and I'm
7 sorry, I wasn't clear on this -- is that we
8 try and make our data freely accessible, and
9 we may try and enable more research to be
10 done in autism.

11 I don't know most of the people
12 who use our resources, but we try and make
13 this, as I said, a learning environment and
14 make people as productive as possible.

15 So it's not surprising that I
16 don't know her as I don't know most of the
17 people who use our resources.

18 Q. Okay. And they're mining your
19 resources researching for the GxE connection,
20 right?

21 MS. BROWN: Objection.
22 Speculation.

23 THE WITNESS: Based on the
24 slides that you've shown here, that is
25 what GEARS -- it looks like. I don't

1 see specific aims, per se, but it does
2 look like they're trying to look for
3 genomics and environmental
4 interactions.

5 QUESTIONS BY MR. TRACEY:

6 **Q. Does SPARK keep track of**
7 **environmental exposures?**

8 A. Can you define what you mean by
9 "keep track of"?

10 **Q. I don't know how else to ask**
11 **that question.**

12 A. Do we have sensors in people's
13 homes? No.

14 **Q. Those are the only two options,**
15 **sensors in people's homes versus --**

16 A. I'm trying, and if you can be
17 more specific about your question, then I can
18 try and answer.

19 **Q. Do you ask about exposures when**
20 **people register with SPARK?**

21 A. It's not a systematic thing
22 that we do. There are people who have used
23 SPARK participants to be able to do those
24 types of studies, and we encourage people to
25 use SPARK in those types of ways.

1 So what I call SPARK central,
2 that has not been our focus, but many other
3 researchers have administered questionnaires
4 and surveys to understand those potential
5 exposures.

6 **Q. So other people, other than you**
7 **as the principal investigator, would have to**
8 **ask the exposure questions because you're not**
9 **doing it yourself in SPARK central?**

10 A. So we set this up as a learning
11 ecosystem --

12 **Q. Okay.**

13 A. -- in which other -- we invite
14 the community, the research community, to be
15 able to think of good questions to ask, and
16 we provide essentially the infrastructure to
17 efficiently ask and gather those data.

18 **Q. No, I understand that.**

19 A. But we allow the community to
20 scientifically drive the questions.

21 **Q. No, I understand that.**

22 **My question, though, is, does**
23 **SPARK central -- is that you, SPARK central?**

24 A. I am the principal
25 investigator, and, yes, I run SPARK

1 centrally.

2 **Q. Does SPARK central ask any**
3 **questions, interrogate or try to figure out**
4 **exposures?**

5 MS. BROWN: Objection to the
6 form.

7 THE WITNESS: Again, the model
8 is that this is meant to be a
9 collaborative scientific model, and so
10 SPARK does the central recruitment to
11 be able to develop and retain and
12 engage the community --

13 QUESTIONS BY MR. TRACEY:

14 **Q. Okay.**

15 A. -- while we allow scientists
16 really to ask the questions.

17 **Q. I see.**

18 **So SPARK central isn't asking**
19 **any questions?**

20 A. SPARK central provides
21 infrastructure for scientists to be able to
22 ask questions.

23 **Q. Yes.**

24 A. We also provide infrastructure
25 to be able to disseminate the learnings from

1 those scientists back to the community.

2 **Q. Sure.**

3 A. And within this, there are
4 central things that we will develop in terms
5 of genomic datasets that we will make freely
6 available to anyone in the community to be
7 able to analyze the data.

8 **Q. What I'm -- I appreciate that.**
9 **It's becoming more clear.**

10 What I was wondering is when
11 somebody -- I think you call it enroll in
12 SPARK, right? That's what I've seen.

13 A. Uh-huh.

14 **Q. When a -- a child or parents**
15 **enroll, right, and even adults, but you don't**
16 **have very many of those, but when they**
17 **enroll, do they answer any questions when**
18 **they enroll that would include prenatal**
19 **exposure questions?**

20 A. For the most part, the
21 questions that we ask are questions related
22 to if the diagnosis was made, confirming the
23 diagnosis, being certain of the autism
24 diagnosis, asking questions about things like
25 medications that are used for the treatment

1 of autism and issues related to family
2 members so that we know who else within the
3 family to invite to be part of SPARK.

4 **Q. Okay.**

5 A. We focus on those types of
6 questions.

7 And then we do have other
8 things that we can ask clinical sites to be
9 able to help provide data.

10 So, as an example, if we have
11 imaging data like brain MRIs or EEGs that
12 family would not have directly access to
13 themselves but clinical sites would have,
14 then they help to enrich with the family's
15 permission those additional very granular
16 clinical data.

17 **Q. Okay. So I didn't hear you say**
18 **that SPARK central asked any questions about**
19 **exposure.**

20 **Is that accurate?**

21 A. For the most part, trying to
22 minimize the burden on families. We are
23 allowing the scientific community to be able
24 to design how and what questions to ask, and
25 we were also letting families decide their

1 level of engagement and the burden for the
2 participation.

3 Q. No, fair. I'm not being
4 critical. I'm just trying to understand it.

5 Okay. So the scientific
6 community then, depending on the willingness
7 of the families involved and their level of
8 engagement, as you said, can ask the
9 questions related to environmental prenatal
10 exposures?

11 A. Correct. And then --

12 Q. Oh, sorry.

13 A. And then those data, as they
14 are collected, become part of the central
15 resource where organically any researchers
16 can ask -- see the data and ask questions
17 themselves and analyze the data, so that
18 those organically can evolve into more and
19 more complex questions and study designs can
20 be addressed.

21 Q. Okay. And do you have -- and
22 then you get access -- you meaning SPARK
23 central. You then have access to the
24 information that the other scientists in the
25 community are putting together when they

1 **engage the families and get the information?**

2 A. So there is a time when those
3 investigators have the data to publish, and
4 then when they publish the data, those data
5 are deposited back in SPARK central for other
6 researchers to be able to use.

7 (Chung Exhibit 320 marked for
8 identification.)

9 QUESTIONS BY MR. TRACEY:

10 Q. Okay. This is Exhibit 320,
11 please.

12 This is from your website. You
13 can see it's SPARK.

14 Do you see that? This is from
15 two weeks ago. Yeah, two weeks ago almost
16 exactly.

17 There we go. Oh, no, it's from
18 three days ago again.

19 SPARK for Autism. That's you,
20 right?

21 A. Yeah.

22 MS. BROWN: Hang on one sec.

23 We're just getting our papers
24 together. Do you have that?

25 Oh, is this just the title of

1 your folder?

2 MR. TRACEY: Yeah.

3 MS. BROWN: Okay.

4 MR. TRACEY: You can keep it.

5 MS. BROWN: Thanks.

6 QUESTIONS BY MR. TRACEY:

7 **Q. All right. So this is from**
8 **your website, SPARK, Simons Powering Autism**
9 **Research, right?**

10 A. Correct, that's from the SPARK
11 website.

12 **Q. And do you know who Emily**
13 **Singer is?**

14 A. She's a writer.

15 **Q. Okay. For SPARK?**

16 A. Science, communicator, writer,
17 yes.

18 **Q. But I mean, for you, for SPARK.**

19 A. She works for the Simons
20 Foundation, but...

21 **Q. Okay. You know her?**

22 A. I do know her.

23 **Q. Okay. And she's put up this**
24 **web page, I guess, is the name of it, Study**
25 **Spotlight: GEARS study. And then she writes**

1 that, "Research suggests that environmental
2 factors, such as a parent's age or the
3 mother's infection during pregnancy, can
4 contribute to autism risk, but identifying
5 those factors has been incredibly
6 challenging. Studies often have conflicting
7 results. One might find that exposure to
8 air pollution increases risk for autism,
9 while another finds no such link."

10 By the way, have you ever seen
11 this before today?

12 A. No. I haven't seen this
13 particular blurb on the website, no.

14 Q. Do you spend any time on the
15 website?

16 A. I have a lot of things that I
17 do, so I have a large team that manages many
18 of the details like this.

19 Q. Okay. Who manages the website
20 for you?

21 A. There's a woman by the name of
22 Beverly Robertson.

23 Q. And what is her background?

24 A. She's our communications
25 director.

1 Q. Okay. Does anybody approve the
2 content for scientific accuracy?

3 A. Well, Emily Singer is herself a
4 scientist.

5 Q. Oh. What kind of scientist?

6 A. I don't recall her degree.

7 Q. So she's a scientist.

8 She goes on to say, Emily does,
9 the scientist, "One reason for this
10 discrepancy could be that that environmental
11 and genetic risk factors can interact. In
12 other words, an environmental variable, such
13 as air pollution, might increase autism risk
14 only in people who are genetically
15 susceptible. A classic example of this type
16 of interaction is phenylketonuria, a
17 metabolic condition linked to intellectual
18 disability. Children with the disorder are
19 at risk of brain damage, but only if they eat
20 a high-protein diet. An environmental
21 factor, diet, and a genetic factor, a faulty
22 metabolism gene, interact to cause
23 phenylketonuria. Early screening and a
24 special diet drastically reduce symptoms.
25 All infants in the United States are tested

1 **for this disorder."**

2 **Did Emily -- what she wrote**
3 **there, is all that correct?**

4 A. Yes, all of that's correct.
5 Phenylketonuria is a very specific use case,
6 but, yes.

7 **Q. But if you don't have -- you**
8 **can have the gene, but if you don't have the**
9 **right environment, you won't get sick, right?**

10 MS. BROWN: Objection to the
11 form.

12 THE WITNESS: This is a
13 medical, metabolic diet that we give
14 these children, so just to -- for
15 people who may not realize this, this
16 is not just, like, not eating a steak
17 sandwich. I mean, this is a metabolic
18 diet that is deprived in phenylalanine
19 to be able to avoid the inborn error
20 of metabolism associated with this.

21 QUESTIONS BY MR. TRACEY:

22 **Q. Okay. I agree.**

23 **But what you're doing is**
24 **controlling the diet, right?**

25 A. I mean, there are multiple

1 examples medically where we, as part of our
2 treatment, will either have ways of being
3 able to deplete the body of something that
4 happens, being able to go around a metabolic
5 block, being able to do substrate inhibition.
6 If you want to call that environment because
7 it's anything that's not the genes, yes,
8 that's what we try and do in medicine, is we
9 try and treat people with these conditions.

10 **Q. Well, I mean, it's not me**
11 **wanting to call it that. It's Emily Singer**
12 **from SPARK calling it that. She says it's an**
13 **environmental factor, diet.**

14 **A.** Again, point of clarification,
15 Emily Singer from the Simons Foundation,
16 but --

17 **Q. Oh.**

18 **A.** -- yes, as she's written this,
19 she is writing an example that people may
20 have familiarity with because phenylketonuria
21 is something we include in newborn screening.
22 So something every baby in the United States
23 is screened for.

24 **Q. And it's highly treatable,**
25 **isn't it?**

1 A. It is, if recognized early.

2 Q. Yeah.

3 They go on to say, Emily does,
4 from the Simons Foundation, "Heather Volk and
5 collaborators at Johns Hopkins University in
6 Baltimore aim to explore whether this type of
7 interaction also applies to autism," and then
8 she starts a quote. "'We think that autism
9 can't be explained only by genes or
10 environmental factors alone,' Volk says.
11 "'It's likely a combination of these two
12 things working together.'"

13 Is Heather Volk correct?

14 MS. BROWN: Objection to the
15 form.

16 THE WITNESS: I think Heather
17 Volk is performing GEARS to answer
18 that question. I don't think she has
19 data yet to know the answer.

20 QUESTIONS BY MR. TRACEY:

21 Q. So when she says, "We think
22 that autism can't be explained only by genes
23 or environmental factors alone," you think
24 she's premature in making that statement on
25 your website?

1 A. Well, I think we can go back to
2 data that we've talked about before with the
3 heritability of 80 to 90 percent, the
4 overwhelming majority of the probability is
5 associated with genetics. That number is not
6 100 percent, so there clearly is something
7 nongenetic, but the overwhelming
8 preponderance of risk and the risk that we
9 have strong data to support with replication
10 are genetic factors.

11 **Q. Doctor, heritability includes**
12 **the environment, doesn't it?**

13 MS. BROWN: Objection to the
14 form. Misstates her testimony.

15 THE WITNESS: Not in the way we
16 define heritability, no.

17 QUESTIONS BY MR. TRACEY:

18 **Q. Not in the way you define**
19 **heritability?**

20 A. Not in the way that the genetic
21 scientific community defines heritability.

22 **Q. Can you point me to a source, a**
23 **reference, that defines it your way; that is,**
24 **heritability by definition excludes any**
25 **environmental contribution?**

1 MS. BROWN: Objection to the
2 form.

3 THE WITNESS: Again, as we
4 think about heritability, these are
5 based on twin studies. The most
6 robust of those twin studies include
7 twins who are reared apart. And so in
8 terms of being able to control for the
9 environment, that does the best that
10 we biologically can in terms of
11 keeping the genes constant and the
12 environments even different.

13 QUESTIONS BY MR. TRACEY:

14 Q. Do you remember my question?

15 A. Can you repeat your question?

16 Q. Yeah. I want to look up a
17 site, a source, that says what you said. I
18 want somebody other than Wendy Chung saying
19 what you say. The claim is, as I understand
20 it, that heritability means genes, genetics
21 and only genetics, and ignores the
22 environment. There is no environmental
23 contribution to heritability in your world.

24 I want somebody other than you
25 who is somebody that knows what they're

1 **talking about that will confirm that.**

2 MS. BROWN: Objection to the
3 form of the question.

4 THE WITNESS: So most standard
5 genetic textbooks, human genetic
6 textbooks, will, in fact, say the same
7 thing that I've just said.

8 QUESTIONS BY MR. TRACEY:

9 **Q. Give me the name of one.**

10 **Because I'm going to go -- on a break, I'm**
11 **going to go look it up and I'm going to see**
12 **if it says what you say.**

13 A. So you can probably go to any
14 standard genetic textbooks that will define
15 heritability and will define heritability as
16 I just have.

17 **Q. Give me the name of one.**

18 A. Emery and Rimoin.

19 **Q. Emery and?**

20 A. Emery and Rimoin.

21 **Q. Okay. Can somebody look that**
22 **up? I want to see if it says that.**

23 **Because that's the first time**
24 **I -- I've read a lot of genetics in**
25 **preparation for your deposition, and every**

1 definition of heritability is the one that I
2 read to you at the beginning, which is the --
3 which is the phenotype is the combination of
4 the gene -- of the genes and the environment.

5 A. With all due respect, having
6 spent my career doing this, I think there may
7 be some misunderstanding.

8 Q. Tell me -- so you -- why is
9 it -- I'll tell you what. Let's do this.
10 Bring up her tape. Her tape.

11 I've got a tape of you I want
12 to play for everybody. And we're going to
13 see if we can untangle what it is you're
14 saying.

15 As I understand it, this is --
16 what you're about to see is you speaking
17 about genes and environment.

18 I guess we should give it an
19 exhibit number.

20 MS. BROWN: Yeah, and can you
21 tell us where it's from and the date?

22 MR. TRACEY: It's from her
23 website.

24 MS. BROWN: Her website?

25 MR. TRACEY: SPARK.

1 MS. BROWN: Okay.

2 (Chung Exhibit 300 marked for
3 identification.)

4 (Video played.)

5 MR. TRACEY: All right. That's
6 it.

7 MS. BROWN: And just for the
8 record, would you guys mark the
9 entirety of this PowerPoint as an
10 exhibit?

11 MR. TRACEY: Yeah, I'm going to
12 use it next.

13 MS. BROWN: Okay, great.

14 MR. TRACEY: I've got it
15 marked.

16 MS. BROWN: Great.

17 MR. WATTS: What was the number
18 for the video? Get that on the
19 record.

20 MR. TRACEY: What is the number
21 for the video, Danny?

22 What is it?

23 DANIEL OLIVO: 300.

24 MR. TRACEY: 300. That's
25 Exhibit 300.

1 MS. BROWN: Okay. And do we
2 have a date of the video or just
3 accessible on --

4 MR. TRACEY: I'm going to get
5 to that. Give me a minute. Give me a
6 minute.

7 MS. BROWN: Okay. I just want
8 to make --

9 MR. TRACEY: I've just begun.

10 MS. BROWN: -- a clear, clear
11 record.

12 MR. TRACEY: Can you -- back up
13 to the -- to the actual -- to the last
14 slide on this. Yeah, just back it up,
15 like, 30 seconds.

16 QUESTIONS BY MR. TRACEY:

17 Q. Now, here's my question to you.
18 That's you, isn't it, speaking?

19 A. That is me.

20 Q. Does Wendy Chung, this Wendy
21 Chung today, Dr. Wendy Chung, agree with what
22 that Wendy Chung said?

23 A. I'll state again, which is what
24 I was trying to say within this video, aimed
25 at a lay audience to be able to make things

1 simple, is that it's complicated. And
2 genetics are a strong factor. As I've shown
3 here and as I say in the video and throughout
4 the rest of the video, genetics are a very
5 strong factor in this, in fact, the
6 overwhelming contributor in terms of
7 probability, but it's not 100 percent. That
8 heritability is not 100 percent; it's 80 to
9 90 percent. And so there are nongenetic
10 factors, and I've demonstrated what people
11 have said were associated features in the
12 past.

13 At the end of the day, it will
14 require a complex analysis, which includes
15 being able to control for genetic factors to
16 understand which of these nongenetic factors
17 are at play.

18 **Q. Can you answer my question?**

19 **A. Can you repeat your question?**

20 **Q. Yeah.**

21 **Are all of the things that we**
22 **just played for the jury of Wendy Chung on**
23 **this PowerPoint, this presentation, do you**
24 **agree with every word you said when you said**
25 **them?**

1 MS. BROWN: Objection. Asked
2 and answered.

3 THE WITNESS: So, again, being
4 able --

5 QUESTIONS BY MR. TRACEY:

6 Q. There's no again. You didn't
7 tell me.

8 MS. BROWN: No, no, no. She --

9 MR. TRACEY: You have to tell
10 me whether you agree with them.

11 MS. BROWN: No, sir. She has
12 to answer your question truthfully --

13 MR. TRACEY: No, no, no. No,
14 no. We're not playing that game.

15 MS. BROWN: I'm sorry, you
16 cannot cut her off.

17 MR. TRACEY: We're not playing
18 that game.

19 MS. BROWN: Okay. She's --

20 MR. TRACEY: I'm entitled to --

21 MS. BROWN: -- going to answer
22 your question.

23 MR. TRACEY: Look, I'm entitled
24 to an answer to the question.
25

1 QUESTIONS BY MR. TRACEY:

2 Q. Do you agree with what you said
3 or not?

4 MS. BROWN: And you can answer
5 that in any way you see fit.

6 MR. TRACEY: Well, you've got
7 to answer it.

8 MS. BROWN: You don't -- she is
9 going to.

10 MR. TRACEY: No, you're --
11 you're --

12 MS. BROWN: You're
13 interrupting.

14 Wait. That's --

15 MR. TRACEY: I'm entitled to an
16 answer.

17 MS. BROWN: First of all,
18 that's not professional or
19 appropriate.

20 Second of all, please allow her
21 to answer the question and you can
22 follow up.

23 Go ahead, Dr. Chung.

24 MR. TRACEY: I was trying to
25 follow up. I just don't want the same

1 answer all over again. I heard ya.

2 MS. BROWN: But you're going to
3 get whatever answer she gives you.

4 MR. TRACEY: Okay.

5 MS. BROWN: Go ahead.

6 THE WITNESS: Okay. So, again,
7 autism is complicated.

8 QUESTIONS BY MR. TRACEY:

9 **Q. I don't care about that.**

10 MS. BROWN: No, no, no.

11 MR. TRACEY: I've heard that a
12 hundred times today.

13 MS. BROWN: No, no, no. No,
14 sir. You need to let her finish.

15 QUESTIONS BY MR. TRACEY:

16 **Q. Let's do it this way.**

17 **I want to know whether any of**
18 **those words that we just played for the jury,**
19 **whether any of those words you want to take**
20 **back today, or do you stand by them?**

21 **A. I stand by we have created and**
22 **I have created SPARK to address what are**
23 **extreme complexities for a very complex**
24 **condition. To be able to get at the**
25 **underlying truth about what increases risk**

1 for autism is going to take large numbers of
2 individuals where we have multiple streams of
3 data to be able to understand this complexity
4 of etiology.

5 Q. Let me ask you this. Nowhere
6 in that four-minute talk section of this
7 PowerPoint, that presentation, did you claim
8 that genetics was overwhelmingly the cause of
9 autism, did you?

10 A. We would have to go through the
11 entirety of my presentation for me to recall
12 exactly what I stated about the genetics, but
13 my recollection is that I did refer
14 repeatedly to the fact that genes were a
15 very, very important component. And I'm
16 guessing I say the predominant, although I
17 don't know if I gave specific numbers.

18 Q. Okay. Did you prepare this
19 slide that's on the screen right now that's
20 called Causes of Autism?

21 A. Collectively, the SPARK team
22 put the slides --

23 Q. And what you have is you have
24 two circles, don't you?

25 A. Yes.

1 Q. The circles are the same size,
2 aren't they?

3 A. They happen to be the same
4 size, but that's not to represent the
5 contributions of the two.

6 Q. Well, that's certainly what
7 somebody would get from it, isn't it?

8 A. I don't know what they would
9 get from it.

10 Q. Okay. On the left, you have
11 genetic factors, and you have inherited and
12 de novo, right?

13 A. Yes.

14 Q. And on the right, you have
15 environmental factors, and those two circles
16 of equal size intersect, don't they?

17 A. Yes.

18 Q. What you're saying here is the
19 causes of autism, and what you said on the
20 part of the tape we just played, is that
21 autism is a complex interaction -- I wrote it
22 down -- "a complex interaction of genes and
23 environment."

24 That's what you said, right?

25 A. Yes.

1 **Q. That's true, isn't it?**

2 A. It depends on the individual,
3 and it depends --

4 **Q. Of course.**

5 A. -- on the contributing factors
6 for each individual.

7 **Q. Of course it does.**

8 A. For instance, environmental
9 factors, nongenetic factors, can include
10 prematurity as an example. And within this,
11 to show you the complexity, there are many
12 factors that can contribute to this.

13 You'll also note that I do not
14 have acetaminophen listed on this, nor do I
15 refer to acetaminophen in the talk that I
16 gave.

17 **Q. I did notice that. I'm going**
18 **to come -- I'm going to get to that in a**
19 **second.**

20 But you don't mention
21 prematurity under environmental factors.
22 That's not one of the factors you list for
23 this talk, is it?

24 A. I did not attempt to go
25 exhaustively through every possible thing,

1 including prematurity. I was trying to refer
2 to that when I was talking about the
3 different life stages and, again, for a lay
4 audience, trying to make this relatively
5 simple.

6 Q. Okay. But what you did put
7 on -- and this is years before you were hired
8 by the lawyers for Johnson & Johnson, right?

9 A. Can you remind us what -- when
10 this talk was given?

11 Q. I think it was 2017, although I
12 have a PowerPoint I'm going to use with you
13 that has the exact same slide from this year.

14 A. Okay. So if this was 2017,
15 this was while we were still evolving in
16 terms of some of our understanding of this
17 complexity.

18 Q. Okay.

19 MS. BROWN: And just for the
20 record, do we have a date of the video
21 that we can put on the record?

22 MR. TRACEY: It's on her
23 website. You can go look at it, I
24 mean.

25 MS. BROWN: Okay. But I --

1 just the people who read this
2 transcript aren't going to be
3 looking at the --

4 MR. TRACEY: Somebody pull it
5 up. I want to say it's 2017.

6 MS. WATTS: It's going to be
7 March '17.

8 MR. TRACEY: What is it?

9 MS. BROWN: Thank you.

10 MS. WATTS: I can confirm it's
11 2017.

12 MR. TRACEY: Okay. 2017.

13 MS. BROWN: Thank you.

14 QUESTIONS BY MR. TRACEY:

15 Q. All right. What you do have on
16 your PowerPoint is on causes of autism on the
17 right, environmental factors, you do have
18 in utero exposure to valproic acid?

19 A. So those were associations that
20 were published as of the time of this talk.

21 Q. Yes.

22 Did you believe it?

23 A. At that point, specifically at
24 the time that we had that, those were the
25 best studies at the time.

1 Since then, there have been
2 improvements in terms of some of the
3 epidemiological studies that have been done
4 to do -- to go back and readdress the same
5 questions, oftentimes adding complexity to
6 the studies.

7 **Q. Are you aware of evidence that**
8 **exonerates valproic acid as a teratogen now?**
9 **Is there something that's changed? That's**
10 **what you're implying?**

11 MS. BROWN: Objection to the
12 form of the question.

13 THE WITNESS: Again, as we've
14 talked about, I haven't recently done
15 a deep dive in terms of reviewing all
16 of the evidence for valproic acid.

17 (Chung Exhibit 315 marked for
18 identification.)

19 QUESTIONS BY MR. TRACEY:

20 **Q. I'll tell you what. Let's do**
21 **this. Give me Exhibit 315.**

22 **Here's a PowerPoint of yours,**
23 **Dr. Chung, from April 2015 --**

24 (Audio interference.)

25 Just so we don't get confused

1 about dates, this is a PowerPoint of yours
2 with your name on it from April of this year,
3 and you're going to see the exact same
4 slides?

5 Okay?

6 MS. BROWN: In fairness, we
7 don't know if it's the exact same
8 slides because we don't have those
9 slides.

10 MR. TRACEY: Yeah. I'm about
11 to hand them to you, and if I'm wrong,
12 I'm certain Dr. Chung will tell me I'm
13 wrong.

14 QUESTIONS BY MR. TRACEY:

15 Q. So this is exhibit -- what did
16 I say, 315? First page.

17 You recognize this as a
18 PowerPoint you gave, "SPARK and The Future of
19 Autism Research"?

20 A. Yes, I do recognize the title
21 slide.

22 Q. And that of course has your
23 name on it, MD, Ph.D., April 25, 2023, just a
24 few months ago?

25 A. Yes.

1 Q. And so if we flip over, there's
2 a slide -- I'm sorry, this is not numbered
3 either. It's the one that says, "Autism is
4 complex. It's not a single condition, and
5 many individuals have related challenges."

6 Oh, there we go.

7 Do you see that slide?

8 A. I do.

9 Q. Did you prepare that slide?

10 A. I and my team did, yes.

11 Q. And you've got "autism spectrum
12 disorder," and then you have a variety of
13 other conditions associated with autism
14 spectrum disorder.

15 Is that right?

16 A. That's correct.

17 Q. And of course we see ADHD up
18 there on the left-hand side at the top, don't
19 we?

20 A. Yes.

21 Q. All right. And then if we go
22 to -- it may be the slide before, but it's
23 "There are many causes of autism."

24 It's actually a slightly
25 different slide in terms of the language, but

1 **it's this slide, "There are many causes of**
2 **autism, but for most individuals we do not**
3 **yet know the cause."**

4 MR. TRACEY: Mike, do you got
5 this one? It may be the one -- the
6 slide before.

7 MICHAEL KAUFFMANN: I'm trying
8 to get to it.

9 QUESTIONS BY MR. TRACEY:

10 Q. Okay. Do you have it,
11 **Dr. Chung?**

12 A. I do.

13 Q. Oh, there we go.

14 So this is your slide from this
15 **year, right?**

16 A. Yes.

17 Q. You say, "There are many causes
18 of autism, but for most individuals, we do
19 not yet know the cause," right?

20 A. That's correct.

21 Q. You don't say the only cause is
22 **genetics, do you?**

23 A. No, I do not.

24 Q. You don't say the predominant,
25 **overwhelming cause of autism is genetics, do**

1 **you?**

2 A. Again, without the audio, I
3 can't recall exactly what my words were.

4 **Q. Well, your -- those words are**
5 **not on this page?**

6 A. Again, you know, I said words
7 over this, and I don't recall what I actually
8 said when I gave the talk.

9 **Q. Well, what you actually wrote**
10 **is, "There are many causes of autism."**

11 **Correct?**

12 A. There are many causes of
13 autism.

14 **Q. You didn't say there's only one**
15 **cause, and it's genes, right?**

16 A. Again, I don't recall what I
17 actually said is the text over for this, but
18 what I traditionally say when I give public
19 talks is that genetics are the overwhelming
20 contributor to what we know of causes of
21 autism or contributors to autism.

22 **Q. Well, if that's true, why not**
23 **put it in the slide so there's no confusion?**

24 MS. BROWN: Objection to the
25 form.

1 THE WITNESS: You can't put
2 everything in a slide, and so you
3 speak as you're giving the talk with
4 the slides.

5 QUESTIONS BY MR. TRACEY:

6 Q. That seems like a nontrivial
7 thing?

8 MS. BROWN: Objection to the
9 form.

10 THE WITNESS: I'm sorry, what
11 is nontrivial?

12 QUESTIONS BY MR. TRACEY:

13 Q. It seems nontrivial to make the
14 claim as you do, that the overwhelming
15 majority of autism cases are caused by
16 genetics and genetics alone?

17 MS. BROWN: Objection to the
18 form.

19 THE WITNESS: I think it's
20 stated very clearly here in terms of
21 the genetic factors, and knowing the
22 different types of genetic factors, I
23 think that is clear.

24 QUESTIONS BY MR. TRACEY:

25 Q. Okay. Well, let's read it and

1 **see how clear it is.**

2 **You wrote, "It's a mix of**
3 **genetic and environmental factors that are**
4 **unknown."**

5 **That's what you wrote, right?**

6 A. Yes.

7 **Q. Is that true?**

8 A. I think we've been through this
9 a few times today. Again, 80 to 90 percent
10 in terms of heritability, with that not being
11 100 percent.

12 There are -- there are factors
13 that are not genetic, but the overwhelming
14 contributor is genetic in etiology.

15 **Q. And yet, those words are**
16 **nowhere to be found, are they?**

17 MS. BROWN: Objection. Asked
18 and answered.

19 THE WITNESS: Again, I don't
20 recall what I said exactly when I was
21 giving these slides, and if I quoted
22 numbers -- and it depends on the
23 audience where I say this and how much
24 I'm trying to overwhelm or not
25 overwhelm an audience with numbers and

1 to make concepts simple.

2 And as I was trying to speak to
3 a lay audience with this, the concept
4 is that this is largely genetic, as
5 you can see here, and --

6 QUESTIONS BY MR. TRACEY:

7 **Q. How can I see that here?**

8 MS. BROWN: Wait, please let
9 her finish.

10 QUESTIONS BY MR. TRACEY:

11 **Q. How can I see with this slide**
12 **that this is largely genetic?**

13 A. I don't recall, again, the
14 words I used during the talk that I gave, but
15 with this being a genetic study, people came
16 into this understanding that genetics were an
17 important part of autism and that we were
18 exploring genetic factors.

19 **Q. Okay. But where do I see on**
20 **this slide, point it out to the judge and the**
21 **jury, where it says, "This is really mostly**
22 **genetic"?**

23 **Where do I find that on the**
24 **slide?**

25 MS. BROWN: Do you have the

1 audio because she's asked several
2 times for the audio?

3 MR. TRACEY: I don't have the
4 audio. I don't. I don't think there
5 is one.

6 MS. BROWN: Well, she's already
7 answered this question.

8 MR. TRACEY: I don't think --
9 no, she hasn't.

10 QUESTIONS BY MR. TRACEY:

11 **Q. Point the words on the page**
12 **that tell me that your claim -- that whenever**
13 **you did this, is that autism is mostly or**
14 **predominantly genetic?**

15 MS. BROWN: I object. It's
16 been asked and answered.

17 Go ahead, Doctor.

18 THE WITNESS: Without having my
19 actual talk, I don't recall what I was
20 saying during the time that I gave
21 this --

22 QUESTIONS BY MR. TRACEY:

23 **Q. I didn't ask you about the**
24 **talk, Doctor.**

25 MS. BROWN: Please let her

1 finish.

2 QUESTIONS BY MR. TRACEY:

3 Q. I'm asking about the words on
4 the page.

5 A. The simple answer to your very
6 specific question is I don't have numbers on
7 the page. I don't recall at the time I gave
8 the talk whether I quoted numbers or not.

9 Whether or not I quoted
10 specific numbers for the purpose of this talk
11 does not change my opinion, however, which is
12 a very specific scientific opinion for a very
13 specific audience in terms of trying to
14 understand etiology of autism.

15 And for that, I need to be
16 quantifiable, and for that I will say, again,
17 that the genetics are 80, 90 percent of the
18 heritability.

19 Q. You're right, but we're going
20 to get back to heritability.

21 I just found a definition of
22 heritability, and let me read it to you and
23 see if you agree with it. I don't think you
24 will.

25 It says, "Heritability does not

1 **indicate what proportion of a trait is**
2 **determined by genes and what proportion is**
3 **determined by environment."**

4 A. Can you tell me what you're
5 reading from, please?

6 Q. **It's not.**

7 Well, hold on. I want to know
8 **whether you agree with it first.**

9 MS. BROWN: Well, we object.
10 We don't know what you're reading
11 from.

12 QUESTIONS BY MR. TRACEY:

13 Q. **It doesn't matter. Do you**
14 **agree with the words that I'm saying out**
15 **loud?**

16 A. I would like to understand the
17 source that you're reading from, please.

18 Q. **Does the truth of the matter**
19 **depend to you upon what the source is?**

20 A. I'd still like to hear the
21 source that you're reading from.

22 Q. **Okay. Well, you don't get to**
23 **know the source. I get to ask the questions;**
24 **you don't, Dr. Chung.**

25 MS. BROWN: Okay. Let's keep

1 it professional.

2 QUESTIONS BY MR. TRACEY:

3 **Q. Here's my question, do you**
4 **agree that heritability does not indicate**
5 **what proportion of a trait is determined by**
6 **genes and what proportion is determined by**
7 **environment?**

8 MS. BROWN: Objection to the
9 form of the question.

10 You can answer, Dr. Chung.

11 THE WITNESS: Again, the
12 heritability estimates are the
13 percentage of the variants of a
14 phenotypic trait that are due to
15 genetic factors, to heritable factors.

16 QUESTIONS BY MR. TRACEY:

17 **Q. So you disagree with this?**

18 A. Again, I've told you what my
19 definition of heritability is.

20 **Q. Okay. But I'm asking you a**
21 **follow-up question because it means that you**
22 **disagree with this definition of**
23 **"heritable" --**

24 MS. BROWN: Objection.
25

1 QUESTIONS BY MR. TRACEY:

2 Q. -- "heritability."

3 MS. BROWN: To the form. Asked
4 and answered.

5 QUESTIONS BY MR. TRACEY:

6 Q. Is that right, Doctor?

7 A. Again, in terms of
8 heritability, it's the fraction of the
9 phenotypic variance that are due to heritable
10 factors.

11 Q. And it is -- there's no
12 equation that you're aware of that factors in
13 the environment -- environmental exposures?

14 MS. BROWN: Objection. Vague.

15 QUESTIONS BY MR. TRACEY:

16 Q. That's what you're saying, the
17 pheno -- phenotype is only genetics and no E?
18 All G, no E?

19 A. That is not what I said.

20 Q. Okay. Un-confuse me. I
21 thought you just said that heritability
22 was the -- was a -- was the contribution of
23 genetics to a phenotype.

24 A. What I believe I said, although
25 you can read back what I said to make sure,

1 was the fraction of the variance of the
2 phenotype that was responsible or due to
3 heritable factors.

4 Q. Okay. But phenotype
5 includes -- those are traits that would
6 include and subsume environmental exposures?

7 A. A phenotype doesn't care about
8 genes or environment --

9 Q. It's agnostic.

10 A. -- it is a phenotype.

11 Q. Yeah. Yeah.

12 But it includes -- I mean,
13 Dr. Chung, we don't exist outside of our
14 environment, do we?

15 A. We live in an environment.

16 Q. Yes.

17 Genes don't exist by themselves
18 in a bubble. They're always, always in the
19 confines of environment -- of an environment,
20 right?

21 A. Yes, we are living beings on
22 the earth.

23 Q. Yes.

24 And the environment that we
25 live in impacts us, right?

1 A. It depends on what phenotype
2 one is looking at.

3 **Q. Let me ask it this way.**

4 **Is it your opinion -- are you**
5 **going to testify -- are you testifying that**
6 **phenotypes only include genetic**
7 **contributions?**

8 MS. BROWN: Objection. Asked
9 and answered.

10 THE WITNESS: It depends.
11 You've asked a very general question
12 about phenotypes --

13 QUESTIONS BY MR. TRACEY:

14 **Q. Okay. Let me be more specific.**

15 MS. BROWN: Please let her
16 answer. Please --

17 MR. TRACEY: Hold on. She
18 objected. I'm sustaining her
19 objection.

20 MS. BROWN: I'm sorry, you just
21 keep cutting her off. Please let her
22 finish.

23 QUESTIONS BY MR. TRACEY:

24 **Q. So let me ask a more specific**
25 **question.**

1 Is it your testimony that the
2 phenotype of autism spectrum disorder, that
3 that by definition only includes genetic
4 contributions?

5 A. I believe you've asked that
6 question several times today, and I'll say
7 the same answer again, which is, that I have
8 not said ever today that autism, as a
9 phenotype, is only and exclusively dependent
10 on genes.

11 Q. Let me ask it this way --

12 A. However --

13 Q. I'm sorry. I'll let you
14 finish.

15 A. However, I have repeatedly said
16 that the major contributor is genetics, and I
17 will say that this has been reproducibly in
18 quite rigorous ways.

19 Q. Do you think the heritability
20 definition is different depending on what
21 trait we're talking about?

22 MS. BROWN: Objection to the
23 form of the question. I don't
24 understand it.

25 If you understand it, you can

1 answer.

2 THE WITNESS: Can you define
3 "trait" for me?

4 QUESTIONS BY MR. TRACEY:

5 **Q. No, I can't. Trait -- I mean,**
6 **if you don't know what a trait is, I don't**
7 **know what to do.**

8 A. If you can define "trait" for
9 me, then I can answer the question.

10 **Q. Okay. Do you have a different**
11 **definition of heritability depending on what**
12 **outcome you're looking at?**

13 MS. BROWN: Objection to the
14 form. Vague.

15 THE WITNESS: Again, if you can
16 tell me what it is we're talking
17 about, I feel like you're trying to
18 ask me about any phenotype of any
19 sort, and there's not one single
20 answer for your question.

21 QUESTIONS BY MR. TRACEY:

22 **Q. Yeah, but I don't understand**
23 **how the definition of heritability can be a**
24 **moving target --**

25 A. Can you rephrase --

1 Q. -- and that's what I'm hearing
2 you say.

3 A. Can you rephrase your question?
4 I may not be understanding it.

5 Q. Yeah, I'm not sure that I can.
6 It's your position, as I
7 understand it, that heritability does not
8 include environmental exposures.

9 A. Again, we may have some
10 misunderstandings. A heritability, if it is
11 less than 1, necessarily says that there are
12 environmental exposures that contribute to
13 the phenotype.

14 So the heritability is telling
15 us something about nongenetic factors.

16 Q. Okay. Okay. Is PKU genetic
17 predominantly?

18 A. Phenylketonuria is a monogenic
19 condition.

20 Q. If you don't have the gene, you
21 don't get the disease, right?

22 A. There are multiple monogenic
23 forms of phenylketonuria, but there is not a
24 way to have phenylketonuria without that
25 monogenic condition.

1 **Q. Right.**

2 **And as we've discussed, though,**
3 **if you're not exposed to the environment of a**
4 **diet that will trigger the condition, you**
5 **won't get sick, right? That's what Emily**
6 **Singer told us.**

7 **A. Again, we can pick this apart.**
8 **One will still have phenylketonuria**
9 **regardless of the diet, right. One will**
10 **still --**

11 **Q. That's almost a metaphysical --**
12 **MS. BROWN: Wait.**

13 **QUESTIONS BY MR. TRACEY:**

14 **Q. -- question. I mean --**

15 **A. But one will still have**
16 **phenylketonuria even if we use dietary**
17 **modification.**

18 **Q. Which is why I said you**
19 **wouldn't get sick.**

20 **MS. BROWN: Can you please let**
21 **her finish?**

22 **QUESTIONS BY MR. TRACEY:**

23 **Q. I'm trying to help you out. I**
24 **didn't say phenylketonuria, but --**

25 **MS. BROWN: She doesn't need**

1 help answering your questions.

2 MR. TRACEY: I think she does.

3 MS. BROWN: She can do it all
4 by herself.

5 MR. TRACEY: I'm not sure about
6 that.

7 MS. BROWN: That's not nice or
8 fair or accurate. Please let her
9 finish.

10 THE WITNESS: But we treat
11 people to try and mitigate the
12 symptoms. We're not perfect in our
13 treatment, but we do this to improve
14 health.

15 QUESTIONS BY MR. TRACEY:

16 Q. In 2023, you still have the
17 concentric circles, equal size, overlapping
18 with environmental factors and genetic
19 factors, right?

20 A. For ease of reading this slide,
21 we have these two circles of equal size.

22 Q. And then you have in 2023, just
23 like you did in 2017, you have pollution, mom
24 ill during pregnancy, insufficient folic acid
25 during pregnancy and in utero exposure to

1 **valproic acid.**

2 **That has not changed, has it?**

3 A. Notably, if you compare the two
4 slides from 2023 to, I believe it was 2017,
5 you'll see that I have added mix of genetic
6 and environmental factors are unknown.

7 **Q. Yeah.**

8 A. And that's because, in part,
9 many of those environmental factors on the
10 right we are now revisiting and trying to
11 reanalyze, and it is unknown.

12 **Q. Well, but you could say the**
13 **same thing about genetics then; genetics is**
14 **unknown?**

15 A. Absolutely. We are very
16 rigorously assessing those genetic factors.

17 **Q. Okay. Why did you want to tell**
18 **me that?**

19 A. Why did I want to tell you
20 that?

21 **Q. Yeah.**

22 A. I wanted to make sure you
23 understood that we are trying to apply the
24 most rigorous methods possible to
25 understanding the science and getting forth

1 valuable -- correct information.

2 **Q. How many of the patients in**
3 **SPARK have you identified a monogenic cause**
4 **of autism for?**

5 A. The number is approximately
6 10 percent.

7 **Q. And the other 90 percent, what**
8 **have you attributed their autism to in your**
9 **SPARK study?**

10 A. We're still learning.

11 **Q. The answer is you don't have**
12 **any idea, right?**

13 MS. BROWN: Objection to the
14 form.

15 THE WITNESS: I would not say
16 that it's that we don't have any idea.
17 It is that we're still learning, and
18 to come up with the statistically
19 rigorous information we need for
20 genetics, in particular for genomewide
21 significance, which is a very high
22 bar, we're not willing to declare that
23 we have a known cause except for those
24 10 percent of cases that I referred
25 to.

1 QUESTIONS BY MR. TRACEY:

2 Q. So as you sit here today, the
3 other 90 percent, according to SPARK, are
4 unknown?

5 A. I would say to implicate for
6 any one person the totality of their
7 contributors to their autism, that 90 percent
8 is not yet fully understood.

9 Q. Well, it's unknown, isn't it?

10 A. Or not yet fully understood.

11 Q. What is wrong with using
12 "unknown"? I thought those were your words
13 on the site.

14 A. Because in some cases we know
15 about statistically significant genetic
16 contributors, but we know -- may know that
17 they increase the relative risk by something
18 like tenfold, but that has not met our
19 threshold for things that we can say are
20 cause as a single cause, which I believe was
21 your question.

22 Q. Yeah, we have another clip.

23 MR. TRACEY: What exhibit
24 should we call it?

25 DANIEL OLIVO: This will be

1 391B.

2 MR. TRACEY: And do you have
3 the slide that explains who the guy
4 is? It's another SPARK clip, by the
5 way.

6 DANIEL OLIVO: 391A.

7 (Chung Exhibit 391A marked for
8 identification.)

9 QUESTIONS BY MR. TRACEY:

10 Q. Exhibit 391A. Maybe you can
11 just tell us who this guy is.

12 MS. BROWN: Can we just
13 identify where this is coming from?

14 MR. TRACEY: SPARK.

15 MS. BROWN: And what the date
16 is?

17 DANIEL OLIVO: March 7.

18 MR. TRACEY: March 7.

19 DANIEL OLIVO: 2018.

20 MR. TRACEY: 2018.

21 MS. BROWN: Okay. And both
22 this and the prior video clip are from
23 the SPARK website?

24 MR. TRACEY: Yes, ma'am.

25 (Video played.)

1 MR. TRACEY: There's more.

2 DANIEL OLIVO: 391B.

3 (Chung Exhibit 391B marked for
4 identification.)

5 (Video played.)

6 MR. TRACEY: Stop right there.

7 QUESTIONS BY MR. TRACEY:

8 Q. Did you hear what he just said?

9 A. I heard what he said.

10 Q. Heritability -- they inflate
11 heritability, the twin studies. That's what
12 he said, right?

13 A. That's what Dr. Newschaffer
14 said.

15 Q. Is he a competent scientist?

16 A. I have never evaluated his
17 science to know that.

18 Q. Do you know who he is?

19 A. Broadly, but not in detail.

20 Q. Okay. He calls this a causal
21 pie.

22 Did you hear that?

23 A. I don't recall if he used
24 exactly those terms, but he was talking about
25 gene-by-environment interactions.

1 **Q. And he says at the top,**
2 **"Environmental risk factors do contribute to**
3 **causing autism."**

4 **And you agree with that, right?**

5 A. We've talked about this before.

6 **Q. Yeah.**

7 **Do you believe that the**
8 **heritability in family studies is**
9 **overestimated like this good doctor said?**

10 A. At this point, I still think
11 that heritability estimates are 80 to
12 90 percent. If you look at the science where
13 we have rigorous, reproducible data to
14 implicate many things, the most rigorous, the
15 most reproducible data are for genetic
16 contributions.

17 **Q. Did you -- do you remember my**
18 **question?**

19 A. I do, and I believe I answered
20 your question.

21 **Q. No, you didn't. Let me ask it**
22 **again.**

23 MS. BROWN: She did. She did.

24 She'll do it again.

25

1 QUESTIONS BY MR. TRACEY:

2 Q. Do you believe that the
3 heritability in family studies is
4 overestimated? That's the question.

5 MS. BROWN: Asked and answered.

6 You can answer it again.

7 THE WITNESS: The questions
8 are, I believe, in the heritability
9 estimates of 80 to 90 percent that
10 have been calculated for autism, as
11 well as for ADHD, and those estimates
12 show that the overwhelming
13 contribution is genetic for both of
14 those conditions.

15 MR. TRACEY: I'm going to
16 object to nonresponsive.

17 QUESTIONS BY MR. TRACEY:

18 Q. Do you believe that those
19 studies you just cited are overestimating the
20 heritability?

21 MS. BROWN: Objection. Asked
22 and answered.

23 QUESTIONS BY MR. TRACEY:

24 Q. That's my question. I don't
25 need you to repeat what the studies say. I'm

1 **asking you whether you agree with this doctor**
2 **who believes they're inflated or**
3 **overestimated.**

4 MS. BROWN: So she already
5 answered that. Asked and answered. I
6 object.

7 She can answer it again.

8 THE WITNESS: Again, the
9 heritability estimates for both autism
10 and ADHD are 80 to 90 percent.

11 QUESTIONS BY MR. TRACEY:

12 **Q. I heard that three times.**

13 A. I believe in those estimates.

14 **Q. Are they or are they not?**

15 MS. BROWN: You're cutting her
16 off. I'm going to object.

17 QUESTIONS BY MR. TRACEY:

18 **Q. Are they overestimating?**

19 MS. BROWN: No, no, no, no, no.
20 She's going to finish.

21 THE WITNESS: I believe in
22 those estimates.

23 QUESTIONS BY MR. TRACEY:

24 **Q. So you -- okay. Well, this is**
25 **real simple, Dr. Chung. I'm not a**

1 **complicated person. You either believe**
2 **they're overestimated or you do not. Tell me**
3 **one way or the other.**

4 MS. BROWN: Please don't raise
5 your voice, and please don't
6 interrupt.

7 Go ahead, Dr. Chung.

8 MR. TRACEY: Oh, my God.

9 THE WITNESS: I'll try and say
10 it again.

11 That the heritability estimates
12 that have been published for autism
13 and ADHD of 80 to 90 percent I believe
14 in.

15 QUESTIONS BY MR. TRACEY:

16 **Q. Okay. So you disagree with the**
17 **doctor who was speaking on your website that**
18 **the heritability in the family studies is**
19 **overestimated?**

20 **That's all I'm trying to get**
21 **you to say. You disagree with him.**

22 A. So let me clarify because I
23 realize for understandable reasons that it's
24 confusing about what's on the SPARK website.

25 So SPARK is meant to be a

1 community resource to be able to share
2 information from multiple scientists. It is
3 not endorsed by me in terms of individuals
4 who are speaking on SPARK. We want to
5 encourage discussion so that we can apply
6 rigorous methods to come up with real
7 answers. And to have those --

8 Q. Yeah, I agree. I heard you the
9 first time you told me that.

10 A. -- rigorously validated.

11 Q. I heard you the first time you
12 told me that.

13 A. So within this, I just want it
14 to be clear that I have not personally
15 endorsed his estimates or what he has stated
16 here, and I have stated --

17 Q. I understood that.

18 A. -- what my opinions are.

19 Q. Yeah, I understood that.

20 You disagree with this guy,
21 whoever he is. That's the point I'm trying
22 to make, right?

23 A. That is correct.

24 Q. Okay. I found a quote on
25 heritability in a textbook called Population

1 **Genetics.**

2 **Are you familiar with that?**

3 A. Who are the editors or authors?

4 **Q. Human Population Genetics and**
5 **Genomics.**

6 **And here's the definition.**

7 MS. BROWN: She just asked who
8 the editors are. Do we know?

9 MR. TRACEY: I don't --
10 somebody will tell me in a second. I
11 don't know right this second.

12 And I can't make it bigger.

13 QUESTIONS BY MR. TRACEY:

14 **Q. Charles Reb- -- Alan Templeton,**
15 **Charles Rebstock, Professor Emeritus,**
16 **Department of Biology and Division of**
17 **Statistical Genomics, Washington University,**
18 **St. Louis, Missouri.**

19 A. I'm not familiar with him.

20 **Q. Okay. Well, let's see if he's**
21 **wrong, too.**

22 **He says in his book that**
23 **"Heritability has been misapplied to the**
24 **nature, nurture, genetic environmental**
25 **debate, and even high heritabilities do not**

1 **exclude strong environmental effects. The**
2 **genomic age has allowed measured genotype**
3 **approaches to phenotypic inheritance."**

4 **Is he wrong?**

5 A. I agree with parts of his
6 statement, and I think one has to be very
7 specific about the parts of his statement.

8 Q. Okay. Let's see which ones
9 he's right on. Let me read the first
10 sentence.

11 It says, "Heritability has been
12 misapplied to the nature, nurture, genetic,
13 environmental debate, and even high
14 heritabilities do not exclude strong
15 environmental effects."

16 **Is he right or wrong there?**

17 A. So for conditions like autism
18 that have high heritability, in individual
19 cases there can be nongenetic factors.

20 And for those individual cases,
21 those nongenetic factors may be quite
22 important.

23 Q. Okay. So he's right?

24 A. With the explanation -- I
25 believe in the explanation I gave.

1 **Q. Okay. But --**

2 **A. As an interpretation of what he**
3 **stated.**

4 **Q. Let me read the words again**
5 **because I don't think what you and he are**
6 **saying are inconsistent.**

7 **"Heritability has been**
8 **misapplied to the nature, nurture, genetic,**
9 **environmental debate, and even high**
10 **heritabilities do not exclude strong**
11 **environmental effects."**

12 **You agree with that?**

13 MS. BROWN: Asked and answered.

14 THE WITNESS: Again, when you
15 consider the large number of
16 individuals with autism, so the very
17 high prevalence, to say that there are
18 individual rare cases that could be
19 due to strong environmental or
20 exposure factors, I believe that is
21 likely the case.

22 But the overwhelming majority
23 for individuals are genetic
24 contributors.

25

1 QUESTIONS BY MR. TRACEY:

2 Q. What percentage today -- what
3 percentage of autism today can we attribute
4 to genetic variation -- variant to?

5 A. Can you repeat the question?

6 Q. Yeah.

7 Let me ask you if you agree
8 with this sentence.

9 Well, this disorder, autism, is
10 defined behaviorally rather than genetically
11 or biologically. "At present, a contributing
12 genetic variant can be identified in 5 to
13 30 percent of individuals with ASD depending
14 on the genetic tests used, the cohort
15 examined and the thresholds used for
16 significance." So the range is 5 to
17 30 percent.

18 A. I agree with that statement.
19 I'm not sure you're interpreting the way I
20 am, but I agree with the statement.

21 Q. Okay. You should, because you
22 wrote it.

23 What about this statement?

24 "The incomplete penetrance and variable
25 expressivity of ASD in the presence of a

1 **high-impact genetic variant may be under the**
2 **influence of additional genetic variation,**
3 **including common genetic variation and/or**
4 **epigenetic factors and possibly environmental**
5 **contributions."**

6 A. Can you tell me what that was
7 taken from and when it was written?

8 Q. **It was written this year by**
9 **you.**

10 A. Okay. What I was likely
11 referring to, although I don't have the
12 context of the publication, is that many of
13 the monogenic factors that we have that are
14 associated with autism are always associated
15 with an effect on the brain and behavior, but
16 whether or not an individual has autism, per
17 se, whether or not they meet diagnostic
18 criteria within ADOS and ADI, that's where
19 the incomplete penetrance comes.

20 Q. **Okay. I misspoke, it was 2020,**
21 **in a paper you published in Nature Review**
22 **Genetics.**

23 A. My opinion is still the same as
24 I just stated.

25 Q. **Yeah, I thought it might be. I**

1 **just wanted to correct the record.**

2 **And you go on to say, "A**
3 **polygenic risk score, which expresses the**
4 **cumulative impact of thousands of common**
5 **variants on the probability of a phenotypic**
6 **outcome for ASD, explains approximately**
7 **2.5 percent of the observed variants and has,**
8 **therefore, no clinical use as a risk**
9 **predictor tool" -- "prediction tool in the**
10 **general population."**

11 MS. BROWN: Counsel, if we want
12 to talk in detail about this paper,
13 could she have a copy of it?

14 MR. TRACEY: Yes.

15 MS. BROWN: Thank you.

16 (Chung Exhibit 343B marked for
17 identification.)

18 QUESTIONS BY MR. TRACEY:

19 **Q. Do you agree with that**
20 **statement you made in 2020?**

21 **And this is Exhibit 343B. Do**
22 **you agree with that statement, that --**

23 **A. I'll take just a second so I**
24 **can see the context in which this was**
25 **written.**

1 Q. Yeah.

2 You're talking about polygenic
3 risk scores.

4 A. And can you show me where we're
5 reading?

6 Q. Yeah, top of page 7, the
7 incomplete penetrance sentence is the first
8 full sentence, and then the polygenic risk
9 score sentence is directly following. So I
10 was on the -- I'll just read it again into
11 the record to orient us.

12 "A polygenic risk score, which
13 expresses the cumulative impact of thousands
14 of common variants on the probability of a
15 phenotypic outcome, for ASD explains
16 approximately 2.5 percent of the observed
17 variance and has, therefore, no clinical use
18 as a risk prediction tool in the general
19 population at this time."

20 MS. BROWN: And, Dr. Chung,
21 take as long as you need to
22 refamiliarize yourself with this
23 before you answer.

24 QUESTIONS BY MR. TRACEY:

25 Q. You recognize this as your

1 **paper, right?**

2 MS. BROWN: Well, let's just
3 give her a minute. She has hundreds
4 of papers.

5 MR. TRACEY: Okay. But she
6 might, but her name is on this one, so
7 I just want to set the record
8 straight, but --

9 MS. BROWN: Her name is on 699
10 other ones, so let's just give her a
11 minute to take -- to refresh and
12 answer your question.

13 THE WITNESS: So what I'm
14 referring to is that we are incomplete
15 in our understanding of the genetic
16 complexity of autism. That's not to
17 state that there are not underlying
18 genetic factors, but that we have not
19 yet identified all of them or know
20 mathematically how to be able to put
21 them together in the appropriate way
22 to understand what that does to the
23 probability of autism.

24 We are very immature in our
25 understanding of the genetics, I will

1 admit, but we are -- have made great
2 strides from where we were 10 or
3 20 years ago.

4 And in terms of progress that
5 has been made in autism research and
6 understanding etiology, we have made
7 infinitely more progress in
8 understanding the genetic factors,
9 validating and reproducing those
10 across multiple cohorts and across
11 multiple investigators.

12 QUESTIONS BY MR. TRACEY:

13 Q. What question are you
14 answering?

15 A. The statement --

16 MS. BROWN: The one you asked.
17 Please let her finish.

18 MR. TRACEY: Well, no. I
19 didn't ask her --

20 MS. BROWN: You have to stop
21 interrupting her.

22 MR. TRACEY: I didn't ask her a
23 question. She just started talking.

24 MS. BROWN: But this is not how
25 it works. She'll answer. You can

1 move to strike and ask a follow-up
2 question --

3 MR. TRACEY: What question is
4 she answering?

5 MS. BROWN: -- but interrupting
6 her with these outbursts --

7 MR. TRACEY: It's not on the
8 record.

9 MS. BROWN: -- are not fair,
10 appropriate or consistent with the
11 federal rules.

12 QUESTIONS BY MR. TRACEY:

13 **Q. Do you know what question**
14 **you're answering?**

15 MS. BROWN: Please finish.

16 It doesn't matter. She's in
17 the middle of it. Let her finish.

18 If you can recall where you
19 were, Dr. Chung, please finish.

20 THE WITNESS: In response to
21 the sentence that you highlighted
22 here --

23 QUESTIONS BY MR. TRACEY:

24 **Q. Right.**

25 A. -- polygenic risk score is a

1 portion of that genetic liability. It is the
2 portion that we understand least well, in
3 part, because we have the least amount of
4 data and because it is the most complex.

5 At this point what we can
6 understand from those multiple genetic
7 factors put together with the appropriate
8 weights is a relatively small portion of the
9 genetic variants.

10 And we were responding as
11 authors to concerns that had been raised that
12 individuals would use preimplantation genetic
13 diagnosis and genetic testing on polygenic
14 risk scores to select embryos to try and
15 select against individuals with autism.

16 And we are pointing out that
17 this is something that cannot reliably be
18 done based on the current information.

19 **Q. You were worried about**
20 **eugenics, weren't you?**

21 **A.** There are reasons that I'm also
22 worried about eugenics.

23 **Q. Yeah.**

24 **But my question wasn't this**
25 **broad, social construct that you answered.**

1 **My question was very specific, and it was**
2 **about your very specific statement.**

3 **You state in this paper, "A**
4 **polygenic risk score which expresses the**
5 **cumulative impact of thousands of common**
6 **variants on the probability of a phenotypic**
7 **outcome, for ASD explains approximately**
8 **2.5 percent of the observed variance and has,**
9 **therefore, no clinical use as a risk**
10 **prediction tool in the general population at**
11 **this time."**

12 **So is that a true statement?**

13 A. At the time when this was
14 written, that was a true statement in terms
15 of the percentage of the observed variance
16 based on what we knew about the polygenic
17 risk scores for autism at that time.

18 **Q. And today, what is the**
19 **percentage?**

20 A. I would have to go back. There
21 are unpublished data to be able to say that.
22 The number is higher, but we still do not
23 understand the vast majority of common or
24 inherited variants for autism. Not to say
25 that they do not exist, but we have not been

1 able to put our finger on the genes or
2 variants yet.

3 Q. Is it true today that polygenic
4 risk scores are clinically useless as risk
5 predictors?

6 MS. BROWN: Objection to the
7 form.

8 THE WITNESS: I think in our
9 research studies, we often use
10 polygenic risk score to account for
11 some fraction of genetic contribution.

12 In terms of clinical
13 application, I would not use a
14 polygenic risk score for autism
15 clinically at this point.

16 QUESTIONS BY MR. TRACEY:

17 Q. But my question was about
18 clinically. I didn't -- that's what I -- I
19 didn't ask you about research.

20 I want to know whether -- and,
21 by the way, they're just pointing out -- I
22 was right the first time. This is a 2023
23 paper, July of 2023, last month.

24 A. Okay.

25 MS. BROWN: So it says 2020 up

1 top.

2 MR. TRACEY: So that's what
3 it -- it's strange.

4 MS. BROWN: It says June 2020.

5 MR. TRACEY: I think that's
6 when it was --

7 MS. BROWN: Ran.

8 MR. TRACEY: Look at it, author
9 manuscript --

10 MS. BROWN: It says "published
11 in final edited form, 2020."

12 MR. TRACEY: Okay. So I was
13 right then.

14 THE WITNESS: I think --

15 MR. TRACEY: What's the -- at
16 the bottom -- at the bottom of the
17 page, it says --

18 MS. BROWN: Yeah.

19 THE WITNESS: The time it was
20 released from PMC, which is a way of
21 public access. It looks like it was
22 July '23.

23 QUESTIONS BY MR. TRACEY:

24 Q. So the public got ahold of this
25 in 2023?

1 A. Anyone could have gotten it,
2 but we made sure it was freely available to
3 the public.

4 Q. Okay. Not a huge deal, but I
5 do want to be accurate, and that is
6 confusing.

7 So is it still true today that
8 polygenic risk scores are clinically of no
9 use as risk predictors?

10 A. I would not use a polygenic
11 risk score for autism clinically today, no.

12 Q. Okay. Now, polygenic risk
13 scores, there's some confusion about what
14 those -- there's some confusion about what
15 those actually mean, aren't they?

16 A. I'm sure someone could be
17 confused about almost anything.

18 Q. There's confusion among the
19 genetic -- among geneticists about what it
20 means, isn't there?

21 A. I don't know. Amongst the
22 individuals I work with, I don't think
23 there's much confusion.

24 Q. Well, let me ask this. Do you
25 think that polygenic risk scores encompass

1 **environmental exposures?**

2 A. Polygenic risk scores are
3 literally genetic factors looked at in
4 isolation and their connection or their
5 association with a phenotype.

6 **Q. Really?**

7 A. There can be things that can be
8 modulators, mediators, other things in
9 between, but strictly the way that these are
10 calculated are to look at the genes and the
11 phenotype.

12 **Q. Well, the modulator and**
13 **mediator part of that sentence is important,**
14 **Doctor, isn't it?**

15 A. When we calculate polygenic
16 risk scores, we calculate literally the
17 beginning of the genes and the ends in terms
18 of the phenotype.

19 **Q. And that encompasses whatever**
20 **exposures that the person's been exposed to?**

21 A. We don't account for the
22 exposures.

23 **Q. You don't account for it, but**
24 **it's there?**

25 A. As you have pointed out, we all

1 are living beings that are exposed.

2 Q. And so the assumption that some
3 people make that polygenic risk scores are
4 only accounting for genetic liability is
5 false?

6 A. The calculation is very simple.
7 It takes genetic polymorphisms at the one end
8 and phenotypes at the other end and does not
9 worry about anything in between and does a
10 calculation between the beginning and the
11 end.

12 Q. That's right.

13 But it's the -- between the
14 beginning and the end, that's the important
15 part, isn't it?

16 MS. BROWN: Objection to the
17 form.

18 THE WITNESS: We are agnostic
19 to the middle. We look at the
20 beginning and the end for the purpose
21 of a calculation of a polygenic risk
22 score.

23 QUESTIONS BY MR. TRACEY:

24 Q. You sure do, but the person's
25 not agnostic but they're living in an

1 **environment?**

2 A. From my understanding of the
3 question you asked, you asked about polygenic
4 risk scores, and I'm explaining how a
5 polygenic risk score is calculated.

6 (Chung Exhibit 322 marked for
7 identification.)

8 QUESTIONS BY MR. TRACEY:

9 Q. Let's look at Exhibit 322. Did
10 we give it to her?

11 This is a paper called "The
12 False Dawn of Polygenic Risk Scores for Human
13 Disease Prediction."

14 Are you familiar with this --
15 this commentary?

16 A. I've not read this paper.

17 Q. Do you know what the history of
18 polygenic risk scores is? Do you know how
19 they came about and how recent they are?

20 A. They came out of genome-wide
21 association studies.

22 Q. Do you know how -- where
23 genome-wide association studies came out of?
24 Do you recall agriculture being the first one
25 to attempt to use them for breeding purposes?

1 A. They certainly have been used
2 in agriculture.

3 Q. If you flip over to page 3 of
4 11, you'll see from GWAS to PRS, GWAS is
5 genomewide association studies, right? And
6 that's how we calculate PRS, right?

7 A. GWAS is genomewide association
8 studies.

9 Q. It says, "The principle of
10 combining information at different genetic
11 markers to construct a genetic score was
12 first introduced by animal geneticists to
13 perform a marker-assisted selection."

14 Did you know that that?

15 A. I'm not an animal breeder or a
16 horticulturist, so I'm not familiar with
17 animal genetics in large part.

18 Q. They go on to say, "This score
19 was later called estimated breeding value and
20 is used to estimate offspring performance but
21 for quantitative traits of agricultural
22 interest.

23 "The principles of EBV are
24 built themselves on the assumption that it's
25 possible to separate the genetic and

1 environmental variance components of a trait.
2 Further -- furthermore, that there are
3 typically large number of genetic variants
4 that each capture a small -- capture a small
5 part of the genetic variance, and the
6 heritability of a trait, thus -- can thus be
7 recovered by summing across their
8 contributions; this being the polygenic model
9 of inheritance proposed by R.A. Fisher."

10 Do you understand that to be
11 how polygenic models were developed?

12 MS. BROWN: And I'll just
13 object to the extent she said she's
14 not familiar with this paper, so if
15 you need time to evaluate it to answer
16 that question, you should do that.

17 THE WITNESS: Within this,
18 again, I am not an animal breeder, and
19 so I'm not going to comment on any of
20 the things that are related to animal
21 breeding.

22 QUESTIONS BY MR. TRACEY:

23 Q. All right. I think we're going
24 to leave animals in the next sentence.

25 It says, "PRS was introduced in

1 the human genetics by Wray, et al., 2007,
2 according to the same principle of summing
3 across the contribution of many different
4 SNPs, but serve a different purpose; that is,
5 predicting the individual risk to develop a
6 disease."

7 Now, do you remember one of the
8 very first articles I used with you with the
9 definition of heritability you didn't agree
10 with was by three authors, Visscher and Wray,
11 and the other one escapes me.

12 But do you remember me asking
13 if you had heard of Wray?

14 A. Yes, I do.

15 Q. You didn't know that Wray was
16 the actually geneticist that introduced PRS
17 into human genetics?

18 A. I don't know whether Wray was
19 or not.

20 Q. Okay. If you flip over to
21 page 5, in the middle of the page, starting
22 with "underlying the PAL model" --

23 A. I'm sorry, what is the PAL
24 model?

25 Q. Oh, it's the polygenic additive

1 **liability model, which is how PRS is**
2 **calculated.**

3 A. If you can give me just a
4 second to look at that.

5 **Q. Yep.**

6 **Are you there?**

7 A. I'm just going back a page.
8 Give me a second, please.

9 **Q. It tells you where the PAL**
10 **model is, the definition on erroneous**
11 **assumptions of PRS on page 4.**

12 MS. BROWN: She just needs a
13 minute. She's not familiar with this
14 paper.

15 MR. TRACEY: Oh.

16 QUESTIONS BY MR. TRACEY:

17 **Q. Are you ready?**

18 A. If you can go back to your
19 question.

20 **Q. Yeah.**

21 So I was just going to -- we're
22 going to read a little bit of that sentence,
23 and then I'm going to ask you if you agree or
24 disagree.

25 It says, "Underlying the PAL

1 model is also the assumption that each
2 environmental factor has a small effect that
3 is independent from the effects of the
4 genetic factors. For most traits, however,
5 this is clearly not true.

6 "The effect of the environment
7 can be important, and a given genotype can
8 react differently across different
9 environments. This is well-known from
10 breeders. Changes in the environment can
11 have a dramatic impact on the efficiency of
12 cattle breeding programs and the accuracy of
13 selection.

14 "This occurs, for example, in
15 the case for milk production, with a
16 significant impact of environmental factors
17 such as temperature and humidity and
18 gene-environment interactions.

19 "The environment cannot be
20 ignored, and as explained by Feldman and
21 Lewontin, 'Partitioning the causes of
22 variation is really illusory. The genetic
23 variance depends on the distribution of
24 environments, and the environmental variance
25 depends on the distribution of genotypes.'"

1 **Do you agree with that sentence**
2 **in -- that I read in quotes?**

3 MS. BROWN: Well, I object
4 based on her prior testimony about
5 animals, so...

6 MR. TRACEY: This is -- she's
7 not referring to animals there.

8 THE WITNESS: So I --

9 MS. BROWN: It's milk
10 production.

11 MR. TRACEY: All right.

12 Let's --

13 QUESTIONS BY MR. TRACEY:

14 **Q. All right. Let's -- let me ask**
15 **you this. Let's pretend for a second -- I'm**
16 **going to prove it in a minute.**

17 **Let's pretend for a second that**
18 **that quote applies to humans.**

19 **Would you agree or disagree**
20 **with that quote, "partitioning the causes of**
21 **variation is really illusory; the genetic**
22 **variance depends on the distribution of**
23 **environments, and the environmental variance**
24 **depends on the distribution of genotypes"?**

25 MS. BROWN: I object. Lacks

1 foundation.

2 THE WITNESS: So all of this
3 depends on the phenotype one is
4 talking about. This can't be stated
5 in a monolithic way across everything
6 in human health or disease or
7 behavior. So this depends on what
8 we're talking about.

9 When it comes to environment,
10 yes, for certain conditions. The
11 environment can change. It can have
12 effects in terms of the expression on
13 human's phenotypes, and that may be in
14 some conditions differential based on
15 genotype.

16 Notably, this is not referring
17 to autism specifically, and even if we
18 are talking about environmental
19 contributions, which I've already
20 talked about regarding autism and
21 ADHD, none of this is in reference to
22 that.

23 And animal breeding data are
24 not relevant to a complex human
25 behavior like autism.

1 QUESTIONS BY MR. TRACEY:

2 Q. Okay. Let's read on and see
3 what they say about humans, Doctor.

4 "In their food steps, Burt
5 claims that, 'the conceptual, biological,
6 model in which heritability studies and thus
7 also PRS depend, that of identifiable
8 separate effects of genes versus the
9 environment on phenotype variance, is
10 unsound.' "

11 Do you agree that it is
12 unsound?

13 A. I will point out that these
14 investigators are commenting specifically on
15 polygenic risk scores, and as we've talked
16 about before when it comes to autism, autism
17 genetics are not solely polygenic risk
18 scores.

19 Q. Well, do you agree with them
20 then?

21 A. They're a complex combination
22 of inherited variants, both common and rare,
23 as well as de novo variants, and the
24 complexity of this, putting all of that
25 together, still argues very strongly that the

1 predominant susceptibility or increased risk
2 for autism and ADHD is genetic.

3 And this has been reproducibly,
4 reliably and statistically, significantly
5 shown multiple times by multiple
6 investigators in multiple cohorts.

7 MR. TRACEY: I'm going to
8 object as nonresponsive.

9 QUESTIONS BY MR. TRACEY:

10 Q. Can you answer my question now?

11 MS. BROWN: She did.

12 THE WITNESS: Again --

13 QUESTIONS BY MR. TRACEY:

14 Q. No, no, not again. I want an
15 answer to my question.

16 MS. BROWN: She answered it.

17 It doesn't matter if you like it.

18 QUESTIONS BY MR. TRACEY:

19 Q. Do you agree or disagree with
20 the highlighted part of this paper?

21 MS. BROWN: Objection. Asked
22 and answered.

23 THE WITNESS: I believe that
24 people, human beings, are complex, and
25 that when one is talking about

1 conditions where there is strong
2 heritability but environmental
3 contributions, these things come
4 together. And within this, the
5 heritability estimates, especially
6 when they are as high as they are with
7 autism -- and you have to understand
8 within the landscape of human
9 behaviors and human conditions, this
10 is at the extremely high end. And
11 with that, it is still very likely the
12 case that genetics are the predominant
13 contributing factor.

14 MR. TRACEY: Objection.

15 Nonresponsive.

16 QUESTIONS BY MR. TRACEY:

17 Q. Can you answer my question,
18 Doctor?

19 MS. BROWN: Asked and answered.
20 Twice.

21 MR. TRACEY: No. No, ma'am.

22 MS. BROWN: Yes, sir.

23 QUESTIONS BY MR. TRACEY:

24 Q. Can you answer my question?
25 You either agree with that statement or you

1 **don't.**

2 A. Again --

3 **Q. No, not again.**

4 MS. BROWN: Please let her
5 answer.

6 QUESTIONS BY MR. TRACEY:

7 **Q. Agree or disagree? Yes or no?**

8 MS. BROWN: She doesn't have to
9 answer yes or no. She's going to
10 answer it truthfully and accurately as
11 she's done twice, and now she'll do a
12 third time.

13 QUESTIONS BY MR. TRACEY:

14 **Q. What's the answer to my**
15 **question, Doctor?**

16 MS. BROWN: She absolutely does
17 not have to answer it yes or no.

18 QUESTIONS BY MR. TRACEY:

19 **Q. Do you agree or disagree with**
20 **him?**

21 A. For the complexity of autism --

22 **Q. I'm not asking about autism.**

23 MS. BROWN: It doesn't matter.
24 You can't interrupt her.

25

1 QUESTIONS BY MR. TRACEY:

2 Q. I didn't ask you about autism.

3 MS. BROWN: It doesn't matter.

4 You can't interrupt her.

5 MR. TRACEY: I didn't ask you

6 about autism. You don't get to

7 reframe my questions.

8 MS. BROWN: But you don't get

9 to interrupt. It's rude, and it's not

10 consistent with the rules.

11 MR. TRACEY: It's not rude.

12 This is rude --

13 MS. BROWN: It is.

14 MR. TRACEY: -- and this is

15 obstructive.

16 MS. BROWN: You are

17 interrupting her repeatedly. You may

18 not like her answer, but it is --

19 QUESTIONS BY MR. TRACEY:

20 Q. Doctor, look at me. Look at me
21 in the eye.

22 MS. BROWN: No, we're not --

23 we're looking wherever she wants to

24 look.

25

1 QUESTIONS BY MR. TRACEY:

2 Q. I am not asking you about
3 autism.

4 A. Can you restate your question?

5 Q. I'm asking you about polygenic
6 risk scores and a statement in this paper.
7 Don't reask it a different way.

8 A. So one cannot generalize to all
9 of phenotypes --

10 Q. Okay.

11 A. -- in this way.

12 Q. All right.

13 A. If you would like to increase
14 the specificity of your question, then I can
15 answer this in a way that is correct
16 scientifically.

17 Q. Okay. Do you know who Moore
18 and Shenk are?

19 A. I don't know.

20 Q. You've never heard of those
21 geneticists?

22 A. I'm not sure who you're
23 referring to.

24 Q. That's my question. You don't
25 know who I'm referring to?

1 A. I don't know who you're
2 referring to.

3 Q. Okay. They go on to say,
4 **"Gottlieb considers that genes are part of a**
5 **'developmental system,' and Moore and Shenk**
6 **explain that contemporary biology has**
7 **demonstrated that traits are the product of**
8 **interactions between genetic and nongenetic**
9 **factors at every point of the development."**

10 Is that true or false?

11 A. Again, I think they are talking
12 in a very general way about biology.

13 Q. Do you think that autistic
14 **patients are independent of human biology?**

15 A. I think you have to get down to
16 the level of specificity of individual
17 conditions to be able to understand the
18 factors, genetic and environmental and
19 developmental, for any one condition. You
20 can't generically speak about this
21 accurately.

22 MS. BROWN: Can we take a break
23 when you get to a good spot?

24 MR. TRACEY: Yeah, let me --
25 I've got a couple more things here.

1 QUESTIONS BY MR. TRACEY:

2 Q. It says GWAS and Causal
3 Inference.

4 Do you see that?

5 A. I see that section.

6 Q. It says, "Underneath PRS is the
7 idea that there are individuals with a
8 genetic makeup that predispose them to
9 developing disease, and it's possible to
10 identify such individuals based on their PRS
11 values."

12 That's the -- what's -- what
13 they want to do, right?

14 A. I'm not sure who you mean
15 "they" and how I can tell if they want to do
16 what they want to do.

17 Q. Well, underneath the PRS is the
18 idea that we can do risk predictions in
19 people, right?

20 A. I believe there are people who
21 would like to be able to predict risk.

22 Q. They say, "The SNPs used in PRS
23 identified by association tests on the basis
24 of different genotype distributions in cases
25 and controls. It is thus assumed that these

1 **observed differences in genotype**
2 **distributions reflect the true genetic**
3 **effect. However, this is not true."**

4 **That's what these authors say,**
5 **don't they?**

6 A. They go on to explain that
7 there can be cases of population
8 stratification that can obscure those cases.

9 And so genomewide association
10 studies have to be very carefully designed
11 for population stratification to see the true
12 effect of a true biological variance to
13 calculate an accurate polygenic risk score.

14 **Q. And the truth is to calculate a**
15 **accurate polygenic risk score, you need to**
16 **know the environmental influences?**

17 A. Again, the way genomewide
18 association studies are done to then
19 calculate a polygenic risk scores are to take
20 the genotypes and the phenotypes and not
21 consider anything else in between. And with
22 that, in that population, they can --
23 those -- if you draw from that same
24 population, that PRS should be able to
25 predict with the limitations of whatever

1 those polymorphisms are that are included.

2 Q. All right. Flip over to
3 page 6. In the middle of the page it says,
4 "Matching cases and controls on all
5 environmental risk, however" -- "is, however,
6 not possible."

7 A. I'm sorry, I can't see where
8 you are yet.

9 Q. It's highlighted. "Matching
10 cases and controls on all environmental risk
11 is, however, not possible.

12 "And controls used in GWAS are
13 usually less exposed to environmental risk
14 factors than the general population. Even in
15 a population considered as genetically
16 homogeneous, environmental stratification may
17 well exist, and PRSs have been shown to be
18 sensitive to changes in variables such as
19 age, sex, economic" -- "socioeconomic status
20 as shown in recent studies of the UK Biobank.
21 Therefore, PRSs not only contain information
22 about genetic factors inherited from parents,
23 but also on clinical and environmental
24 factors. As Janssens rightly pointed out,
25 PRSs do not measure what they are supposed to

1 **measure. This fact is totally ignored by**
2 **those who integrate PRS values in risk models**
3 **as an observation, which is independent from**
4 **clinical and environmental factors."**

5 **Do you agree with that?**

6 A. So this is separate from other
7 things we have discussed.

8 **Q. It is.**

9 A. Polygenic risk scores, in
10 general, are taken, and I use my words
11 carefully, from a population and applied to
12 that same population. Therefore, the
13 assumptions made in the original study will
14 apply to that same population when applied
15 again.

16 **Q. Well, only if they're exposed**
17 **to the same environmental factors?**

18 A. As I said, the same population.

19 **Q. Well, no. The same population**
20 **can have different -- home to home can have**
21 **different environmental factors. In one home**
22 **you might take valproate and another home,**
23 **you may take acetaminophen?**

24 A. Again, population is the key,
25 because these are applied to populations.

1 Q. Oh, I see what you're saying.

2 So you should see -- okay.

3 If you flip over to the
4 Discussion page, because I think you're going
5 to -- you're going to recognize some of this
6 language.

7 On the third paragraph down, it
8 says, "The misinterpretation of PRS has
9 dangerous clinical and eugenic consequences."

10 The eugenic consequences are
11 some of the things you were writing about in
12 your paper. That's what you were worried
13 about, one of the things.

14 Right?

15 A. I worry about eugenics in
16 general.

17 Q. Yeah.

18 Well, I mean -- but it came up
19 in the context of your paper we were reading
20 together, right?

21 A. I will just say that I worry
22 about eugenics in general.

23 Q. Okay. Well, you brought it up
24 when we were reading your paper.

25 What am I doing wrong? What am

1 I saying it wrong?

2 A. I'm just making it clear that I
3 have concern -- I try to be an ethical
4 person, in addition to a scientifically
5 accurate person, and so I try and keep that
6 in mind in terms of concerns about eugenics.

7 Q. Okay. Yeah, I wasn't
8 suggesting you were limiting your concerns
9 about eugenics to one paper.

10 Anyway, in the next sentence
11 down, it says, "Furthermore, after individual
12 medical future selling" -- "future selling by
13 direct-to-consumer companies, new companies
14 are now offering prenatal diagnoses, based on
15 PRS, for all the diseases that parents want
16 to avoid infecting their children. Some
17 already protested from an ethical standpoint
18 against such eugenic drift. However, in
19 addition to the ethical question, one must
20 also be aware that it is scientifically
21 wrong. It is important to stop selling the
22 dream that it is possible to predict disease
23 risk with an oversimplistic model."

24 Do you agree with that?

25 A. I agree with as we've spoken

1 about today that the polygenic risk scores
2 that we have capture a small portion of the
3 genetic factors. They are not in this paper
4 talking anything about rare genetic variants
5 and being able -- and for many individuals,
6 the predominance of the genetic risk or
7 liability is actually a rare variance rather
8 than common variance.

9 Furthermore, the polygenic risk
10 scores that have been calculated today are
11 not capturing all of the common variants
12 either.

13 So as I've said, we still have
14 further information to gather on the
15 genetics, but that doesn't deny the fact that
16 genetics are the strongest and most
17 reproducible contributor to autism
18 probability.

19 **Q. I don't know what your answer**
20 **was.**

21 **Did you agree with them or not?**

22 **A.** As I've said, currently, the
23 models that we have for the amount of
24 information that we know about common
25 variants is incomplete and inadequate to

1 accurately predict human behavior, including
2 autism.

3 However, this is a small
4 fraction of genetic liability and that does
5 not -- by not accounting for those other
6 portions of the genetic contribution, that
7 does not negate the genetics beyond PRS that
8 is currently understood is the major
9 contributor to behavior -- the behavior of
10 autism and ADHD.

11 MR. TRACEY: Okay. I'm going
12 to object to nonresponsive.

13 QUESTIONS BY MR. TRACEY:

14 **Q. That question, this sentence is**
15 **only about PRS, right?**

16 A. I'm clarifying that the current
17 understanding of PRS cannot accurately
18 predict human behavior.

19 **Q. Stop. I agree with you there.**

20 MS. BROWN: Well, okay. But
21 you can't -- you can't interrupt and
22 put your hand up and ask her to stop.

23 MR. TRACEY: But I have to to
24 keep control of this deposition.

25 MS. BROWN: No, no, no. But

1 that is not allowed.

2 MR. TRACEY: It is allowed.

3 MS. BROWN: You just put your
4 hand up and told the witness to stop.

5 MR. TRACEY: I did.

6 MS. BROWN: She was
7 mid-sentence, answering your question.
8 That's not acceptable.

9 Please finish.

10 MR. TRACEY: She was not
11 mid-sentence. If her history is
12 any --

13 MS. BROWN: You put her hand up
14 and told her to stop --

15 MR. TRACEY: -- example, she
16 was at the beginning of a sentence.

17 MS. BROWN: -- as if we're at a
18 traffic light.

19 Please finish, Doctor.

20 QUESTIONS BY MR. TRACEY:

21 **Q. That part you just gave me is**
22 **consistent with what's on the screen, right?**

23 A. I want to explain beyond
24 that --

25 **Q. But I don't want to know about**

1 beyond. I didn't ask about beyond.

2 A. But this is --

3 Q. See, when I ask questions,
4 they're only about very discrete subjects.
5 This is not your time to give a lecture, give
6 a PowerPoint presentation. This fine lady
7 right here, when I'm done, can ask you all of
8 those questions.

9 MS. BROWN: Let's take a break.

10 QUESTIONS BY MR. TRACEY:

11 Q. But I have a limited amount of
12 time, Dr. Chung.

13 You understand that?

14 MS. BROWN: Let's take our --

15 THE WITNESS: I understand.

16 MS. BROWN: Can we take our
17 break now? We've been going for an
18 hour.

19 QUESTIONS BY MR. TRACEY:

20 Q. But I've got to finish this.
21 Did that part -- your last part
22 of that question, when I did say stop, that
23 part of your answer was consistent with what
24 we see on the screen, right?

25 A. It is consistent with a very

1 narrow-focused question about the current
2 state of clinical utility of polygenic risk
3 scores.

4 MR. TRACEY: Yes. All right.
5 Let's take a break.

6 MS. BROWN: Thank you.

7 VIDEOGRAPHER: The time is
8 3:09 p.m., and we're off the record.
9 (Off the record at 3:09 p.m.)

10 VIDEOGRAPHER: The time is
11 3:23 p.m., and we're on the record.
12 (Chung Exhibit 322B marked for
13 identification.)

14 QUESTIONS BY MR. TRACEY:

15 Q. Dr. Chung, I've handed you
16 Exhibit 322B, which is a paper, a guidance
17 paper, by the -- or the ASHG. That's the
18 American Society of Human Genetics; is that
19 right?

20 A. Yes, that is.

21 Q. Do you belong to that society?

22 A. Yes, I do.

23 Q. And this is in their journal,
24 which is called the American Journal of Human
25 Genetics, and this was published in December

1 of 2022, eight months or so ago.

2 A. Yes, I see that publication
3 date.

4 Q. Did you read this guidance
5 document when it came out?

6 A. I skimmed over it, yes.

7 Q. Okay. And it's called
8 "Addressing the challenges of polygenic
9 scores in human research."

10 Isn't it?

11 A. That's the title of the
12 manuscript, yes.

13 Q. Do you know or recognize any of
14 the authors that are listed there?

15 A. Yes, I do.

16 Q. Who do you know?

17 A. Claudia Gonzalo {sic} and Sarah
18 Tishkoff.

19 Q. Okay.

20 A. Gonzaga, I should say.

21 Q. Sorry?

22 A. Claudia Gonzaga and Sarah
23 Tishkoff.

24 Q. Okay. This paper, at the top
25 you see, this is a guidance document, ASHG.

1 **This is a guidance document. And if we look**
2 **down at the introduction on the right-hand**
3 **column, they tell us that this "ASHG guidance**
4 **was developed by a writing group led by**
5 **members of the professional practice and**
6 **social implications committee to discuss and**
7 **provide recommendations on three major issues**
8 **related to the technical, ethical and social**
9 **considerations of PGS. It was approved by**
10 **the ASHG board of directors for publication**
11 **in October of 2022."**

12 **Did you -- by the way, are you**
13 **on the board of directors for the ASHG?**

14 A. Yes, I am.

15 Q. **Did you vote and approve this**
16 **paper?**

17 A. Yes, we did at our board
18 meeting.

19 Q. **And so did you vote with**
20 **approval to publish this document for**
21 **everyone to see?**

22 A. By the rules of our board, I'm
23 not supposed to disclose anything about our
24 board meetings publicly.

25 Q. **Okay. But you were on the**

1 board that voted to approve this. Whether
2 you voted yay or nay is none of my business
3 is what you're saying?

4 A. Respectfully, yes.

5 MS. BROWN: Very politely.

6 QUESTIONS BY MR. TRACEY:

7 Q. Yeah, okay.

8 In any event, you were on the
9 board that did vote?

10 A. Yes.

11 Q. All right. Down below at
12 the -- beneath there it says, "Develop
13 diverse research cohorts and analyses."
14 Well, let me ask you this. I should have
15 asked this.

16 Did you edit this document
17 before it was published?

18 A. No, I did not.

19 Q. Okay. You didn't redline it or
20 offer any contributions?

21 A. That was not our role as the
22 board.

23 Q. Okay. It says, "Develop
24 diverse research and cohorts and analyses.
25 Several limitations of PGSs arise from the

1 lack of diverse cohort data in PGS
2 development. The accuracy of a PGS might be
3 compromised when applied to a cohort that
4 differs in key demographic characteristics
5 from the discovery cohort used to develop the
6 PGS. This is known as the PGS portability
7 problem, sometimes also called the
8 generalizability problem or transferability
9 problem."

10 Did I read all that correctly?

11 A. Yes, you did.

12 Q. Do you agree with all of that?

13 A. Yes, I do.

14 Q. Okay. They say, "Several
15 factors contribute to the portability
16 problem, although the relative importance of
17 each is still an open topic of research.
18 First, they talk about linkage disequilibrium
19 varies across human populations. And because
20 PGSs are based on marker loci that are in LD
21 with as yet unknown causal loci, inaccuracies
22 will be introduced if one assumes the GWAS
23 marker loci have the same LD with causal loci
24 in different populations."

25 What is LD? That's the linkage

1 **disequilibrium?**

2 A. Yes, that's what LD stands for.

3 Q. Did everything I read -- did
4 you agree with everything I just read?

5 A. Yes, I do.

6 Q. Okay. It says, "Fine-mapping
7 approaches that aim to identify causal loci
8 can help ameliorate this problem.

9 "Second, different causal
10 alleles will be at different frequencies or
11 even absent, in different populations. At a
12 global scale, a variant common in one region
13 is more likely to be found in several others,
14 whereas rare variants are more likely to be
15 localized.

16 "Third, even causal variants
17 that are shared might have differing average
18 effect sizes in different populations. One
19 potential cause is differing genetic
20 backgrounds between discovery and application
21 cohorts, which might give rise to different
22 average effect sizes as a result of gene-gene
23 interactions, i.e., epistasis, e.g., see
24 Patel, et al. Another potential cause is a
25 different distribution of nongenetic

1 individual risk factors between the discovery
2 and implementation cohorts when such factors
3 moderate the effect of a genotype. Such
4 factors are varied and phenotype dependent;
5 such examples include age, sex, diet,
6 pollutant, exposure, access to health care
7 and gut microbiome composition. In
8 quantitative and statistical genetics, these
9 factors are often collectively referred to as
10 environment, and they induce what are known
11 as genotype-by-environment interactions or
12 context-independent {sic} effects."

13 MR. WATTS: Context-dependent.

14 QUESTIONS BY MR. TRACEY:

15 Q. Do you agree with all of that?

16 A. So within this, realizing that
17 they were trying to deal with a hard problem,
18 that is to, in a very general and a generic
19 way, to describe PRSs across a wide range of
20 human conditions, phenotypes being that
21 related to health and just normal genetic
22 variation.

23 And what they've written here
24 is quite generic in terms of trying to be
25 broad. As you said, they were trying to

1 provide guidance in a nonspecific way, and
2 the generality with which they refer to this
3 is generally correct.

4 However, to be able to
5 understand how this applies to any one
6 condition, it is necessary to then drill
7 down. The overwhelming concern that they had
8 and that our community continues to have were
9 the items that you addressed at the
10 beginning; and that is that most research
11 studies today are conducted on individuals of
12 European ancestry, and the genetic variation
13 that we understand is largely derived from
14 that genetic ancestry.

15 Some of the technical language
16 that you've read is related to the
17 portability issue that you referred to. So
18 that if we try and port over the information
19 to individuals of African ancestry, Asian
20 ancestry, other parts of the world, the
21 ability to accurately port over that
22 information may be inadequate. And so they
23 are trying to provide general guidance about
24 limitations of PRS, and in particular, some
25 of the pitfalls of why those common variants

1 may differ in their predictive ability across
2 populations around the world.

3 **Q. One of the problems they**
4 **identify in PRS being accurate is the**
5 **different environmental effects in different**
6 **populations?**

7 **A.** They do, but if you look by
8 rank order in terms of order in which they
9 list the issues, and I know this based on
10 being a member of the community, the much
11 greater concern is they're addressing are
12 differences across populations and
13 differences that may be attributable to
14 linkage disequilibrium structure and
15 differences that we see based on that genetic
16 structure, the relative weight and genetic
17 contributions.

18 And in particular, one of the
19 concerns that we've had that's very
20 significant is that if a gene is not
21 implicated in a European population and,
22 therefore, absent in other populations where
23 that gene is applicable, that discovery will
24 not replicate in those other populations.

25 **Q. In the United States, we're**

1 **called the melting pot.**

2 **Are we the melting pot?**

3 A. I would say some regions of the
4 United States more so than others.

5 Q. **Some are more melted than**
6 **others?**

7 A. Some are more admixed and some
8 are more heterogenous, shall we say.

9 Q. **Okay. And I read that**
10 **80 percent or so of the existing discovery**
11 **GWAS cohorts were European, and it would be**
12 **14 percent Asian and a few percent other**
13 **things.**

14 **Is that right?**

15 A. I'm not sure where you read
16 that, but I agree that they are overwhelming
17 of European ancestry.

18 Q. **Okay. And so the portability**
19 **problem, I kind of like that term, it makes**
20 **sense, we can't export this to other**
21 **populations, right?**

22 A. Right.

23 Q. **Now, it would seem to me, if in**
24 **a population -- for example, if you had a**
25 **population that was exposed to something, a**

1 compound, an environmental toxin, and this
2 population had either epigenetic changes or
3 de novo mutations as a result of these
4 exposures, or flipping it around, you're in a
5 population that it was exposed to something
6 that was reducing disease, better diet,
7 things like that, that would also affect the
8 GW -- the GRS {sic} scoring, wouldn't it?

9 A. So you've got several different
10 concepts wrapped within that.

11 Q. Yes.

12 A. So let's try and separate them.
13 I think the fundamental issue
14 in terms of that portability issue is, let's
15 confine ourselves to those common variants.
16 So within the space of genetics, there is a
17 space of common inherited variants, rare
18 inherited variants and de novo variants,
19 right. For simplicity, we'll divide those
20 into three spaces.

21 The polygenic risk scores or
22 the genomewide association studies are only
23 those common genetic variants, and those
24 variants that we use are oftentimes not the
25 causative alleles that are actually causing

1 or attributable. They may be close to. They
2 may travel together and, hence, be in linkage
3 disequilibrium with what the true genetic
4 variance is.

5 Based on the architecture for
6 that population, A and B may travel together
7 if you're European, but A and C may travel
8 together if you're Asian. And so, therefore,
9 by using the surrogate or the substitute, you
10 may get the wrong signal by using A, if it's
11 B or C that are actually causative.

12 So, again, the overwhelming
13 message that's coming through here is that
14 the concern is that people will use
15 discoveries from a population in, say, the
16 2000s, from over here in this part of the
17 world to over here in this part of the world.

18 If one were to take this over
19 here in this part of the world and just move
20 over to the next state, it should be
21 applicable in that other state. Things are
22 common enough within this larger genetic
23 ancestral pool that those should be able to
24 port over.

25 So the large part of what

1 they're concerned about are these really
2 radical differences, but, again, remembering
3 that this is still only a portion of that
4 spectrum of genetic liability.

5 Q. So let me ask you this. This
6 is what I think about. I'm -- I've seen the
7 North Korea, South Korea population mentioned
8 as an example of heritability being a product
9 of environment and genes. And here was the
10 example that was given to me.

11 The South Vietnamese people are
12 of the same genetic architecture as the North
13 Vietnamese people and yet, they're 5 inches
14 taller on average. And we know that height
15 is 80 percent-plus heritable, right?

16 Correct?

17 A. I believe the estimate is very
18 high, as you said.

19 Q. Very high.

20 And so in that environment, if
21 I were doing a PRS for height to see what the
22 risk was for shortness, if you want to say
23 shortness, I would see in the same genetic
24 cohort a stark difference in height that was
25 primarily or exclusively driven by nutrition,

1 **right?**

2 **This is sort of a classic**
3 **example that I've read in the genetics**
4 **literature.**

5 **Are you familiar with that**
6 **example?**

7 A. Right.

8 So as I said earlier, it
9 depends, and the context is incredibly
10 relevant. When it comes to issues of
11 nutrition, right, things that are driven and
12 inextricably correlated with nutrition and
13 access to calories, whether you're going to
14 talk about height or body mass index, but
15 things that are directly related to
16 nutrition, certainly you see differences in
17 terms of those environmental, if you will,
18 factors.

19 But it depends on the context.
20 And so you can't generalize what's true of
21 the condition over here to a condition over
22 here. You always have to remember that
23 specific context.

24 Q. Okay. But I was just -- I
25 **agree with you. I understand.**

1 But generally speaking, the
2 concept, though, is one of in which in the
3 North Viet -- sorry, North Korea, South
4 Korea, you know, cohorts, where the
5 phenotype, shortness or tallness, I don't
6 know what we want -- which one we want to
7 pick, but there's stark differences related
8 not to genetics that a polygenic risk score
9 would imply were genetic, even though they
10 were nutritional?

11 MS. BROWN: Objection to the
12 form.

13 Go ahead.

14 THE WITNESS: So I don't think
15 the polygenic risk score has that
16 implication. Rather, what happens in
17 terms of the calculation of
18 heritability or in terms of polygenic
19 risk tends to be with one population
20 in one moment in time.

21 And so when there are changes
22 in terms of the environment, such as
23 you've described, what you see is you
24 see that same polygenic risk score
25 being able to predict, for instance,

1 the distribution of height and the
2 relative sort of rank order in height
3 that you see, but yet nutrition is
4 expanding for the entire cohort in
5 terms of increasing height.

6 QUESTIONS BY MR. TRACEY:

7 **Q. But if I didn't have access to**
8 **the nutrition information, right, I didn't**
9 **know, I didn't know what the environmental**
10 **differences were, and I just did polygenic**
11 **risk scores against everybody in North and**
12 **South Korea, right? Are you with me?**

13 A. So you're going to put together
14 the entirety of North and South Korea?

15 **Q. All the common variants, I'm**
16 **going to put them all together. And I'm not**
17 **going to have an explanation -- I'm not going**
18 **to have the explanation for the height**
19 **differences in my polygenic risk score, am I?**

20 A. So, again, that's not going to
21 be that driving a factor. What you are
22 pointing out to is the complexity in terms of
23 understanding human phenotypes. And what it
24 drills down to is being able to calculate all
25 the myriad factors that go into calculating

1 and predicting what can ultimately be complex
2 biology; where it's not a one-to-one mapping
3 in terms of one factor or one gene, but,
4 again, it differs depending on what the
5 phenotype is that's involved and how large
6 those environmental contributors are to that
7 factor.

8 Q. Let me give you another
9 example. I like examples. I learn better
10 that way.

11 If we had a population of
12 mothers who did no folic acid
13 supplementation, no prenatal vitamins at all,
14 and we had another population of women that
15 did take them, and we didn't know this, we
16 didn't know that one population was
17 supplementing and the other wasn't
18 supplementing, and they were in the same
19 country, and they were the same -- they were
20 homogeneous, would the outcome differences in
21 autism in those groups be the result of
22 genetics or supplementation?

23 A. So what you're pointing out,
24 though, actually works against you in terms
25 of the predictions of heritability and the

1 role of genetics.

2 If, in fact, your hypothesis is
3 correct and there's a fair amount of
4 environmental influence that varies and is
5 unaccounted for, it will look like you have a
6 weaker genetic signal rather than a stronger
7 genetic signal.

8 So if anything, you've made the
9 argument that we've underestimated the
10 heritability and the genetic contributors.

11 **Q. I'm not following that.**

12 **What I've just described is**
13 **effectively an epidemiology study, which we**
14 **use every day to assess populations. I have**
15 **a control and an exposed group.**

16 A. So taking that example and
17 helping to understand the complexity of these
18 epidemiological studies, many of them
19 heretofore have not included genetics and
20 genetic factors or even simple things like
21 family history.

22 **Q. Yeah.**

23 A. Or maternal factors that may be
24 influencing by transmission without
25 accounting directly for the genetics but

1 factors in terms of enrichment within that
2 family history from either mother or from
3 father.

4 Without accounting for that and
5 accounting in particular why some of those
6 genetic factors may influence directly the
7 exposure, because someone might be driven to
8 use a medication or to have a certain
9 exposure, then, in fact, without accounting
10 for genetics, one could falsely come to the
11 conclusion that it was whatever that
12 environmental exposure was that was driving
13 the cause, but, in fact, it is possible it is
14 the underlying genes. And if one does not
15 account for that, at least indirectly, one
16 can falsely jump to the conclusion that it's
17 the exposure rather than an underlying
18 genetic susceptibility.

19 **Q. So in my example, no folate**
20 **supplementation, none, lots of folate**
21 **supplementation, the one without folate**
22 **supplementation has more children with autism**
23 **in it than the one that had supplementation,**
24 **you think the conclusion to be drawn from**
25 **that is what?**

1 A. So I did not talk about the
2 specificity of folate and autism.

3 **Q. Well, that was my question.**

4 A. So I'm not --

5 **Q. That was my whole example.**

6 A. Okay. So I have not looked
7 carefully at the literature, the
8 epidemiological literature, about folate and
9 autism. So I'm not going to comment on that
10 specific question because I don't know that
11 there are reproducible or reliable studies
12 that have incorporated genetic factors either
13 directly or indirectly with folate and being
14 able to look at reliable outcomes of a
15 phenotype of autism.

16 You would need to have, in my
17 opinion, all of those to be able to come to
18 the most accurate conclusion about
19 understanding contributing factors.

20 **Q. Do you know whether scientists**
21 **have looked at populations of women who don't**
22 **supplement and look at their rates of autism**
23 **compared to those that do?**

24 A. Again, in terms of my opinion
25 today, I didn't prepare to be able to address

1 that question.

2 Q. No, no, I wasn't asking you if
3 you knew the answers. I was asking
4 whether -- if you know that work has been
5 done.

6 A. As I said, I would need to do a
7 comprehensive review to be able to answer
8 knowledgeably about that question.

9 Q. Let's get back to the guidance
10 paper just for a second here.

11 On the same page, next column
12 over, starting with "Given these problems,"
13 we start to see some recommendations.

14 It says, "Given these
15 problems," which -- some of which we've been
16 talking about, "we recommend that future
17 development of PGSSs incorporate increased
18 cohort diversity."

19 That's good, right? That's
20 what you were talking about, right?

21 A. Yes.

22 Q. "Along multiple dimensions,
23 ancestry, age, sex, health care access, and
24 other environmental variables relevant to
25 focal phenotypes."

1 **That's the recommendation,**
2 **right?**

3 A. Again, realizing that this is
4 for a general audience in terms of being
5 inclusive for all phenotypes, yes, that's the
6 recommendation.

7 **Q. Well, this isn't for a general**
8 **audience. This is for geneticists.**

9 A. I'm sorry, let me rephrase. An
10 audience that is nonspecific in terms of the
11 phenotypes under research of geneticists.

12 **Q. Okay. And what the guidance**
13 **document says is we must -- or they recommend**
14 **that we need to take into account other**
15 **environmental problems relevant to the focal**
16 **phenotype.**

17 **A focal phenotype is like**
18 **autism, right?**

19 A. Again, they're being
20 nonspecific within this. And as general
21 guidelines, they're trying to make sure that
22 they're covering all of the bases across
23 different conditions.

24 **Q. But a focal phenotype, an**
25 **example of a focal phenotype is autism?**

1 A. That is correct. If one wanted
2 to study autism, then one could make and
3 tailor these recommendations to autism.

4 Q. They say, "Over the year" --
5 "past few years, several such efforts,
6 including the All of Us, H3Africa and the
7 Trans-Omics for Precision Medicine programs
8 have been launched with the goal of
9 increasing the diversity and representation
10 of previously understudied groups in human
11 genomics research and better understanding
12 the contribution of genetic and environmental
13 factors on disease risks."

14 And you certainly know that to
15 be true, right?

16 A. Yes. And, in fact, the major
17 effort that they've utilized, as they've
18 outlined here, is to increase the diversity
19 of these cohorts to increase the ancestral
20 genetic diversity in particular.

21 Q. And then if you flip over to
22 the next page, they give some more
23 guidelines. They say, "Foster Robustness,
24 Application and Interpretation of PGSSs."

25 And they -- they flag that

1 first, "the accuracy of the effect size
2 estimate used in PGS is dependent on the
3 sample size of the reference GWAS, the
4 genotyping or imputation of accuracy, the
5 provision of trait measurement and GWAS
6 modeling decisions such as those regarding
7 batch-correction and adjustment for
8 covariants.

9 "During estimation of effect
10 sizes in the reference GWAS, a correlation
11 between a variant genotype and unobserved
12 environmental factors," and they say, quote,
13 "Environmental confounding are between a
14 variant genotype and genetic background," and
15 they quote, "Genetic confounding, can lead to
16 inaccurate effect-size estimates."

17 Do you agree with that?

18 A. Again, realizing that this is
19 written generically across all phenotypes,
20 what they're alluding to, again, across many
21 different phenotypes is that the effect size
22 can be misrepresented within a polygenic risk
23 score calculation.

24 Q. Right.

25 A. That's not --

1 **Q. Oh, sorry.**

2 A. That's not to say that genetics
3 as a contributor to the condition are changed
4 in terms of estimates of genetic risk.

5 **Q. Well, but what will be done is**
6 **the environment will be underrepresented?**

7 A. Again, with the complexities of
8 whether these are not moderators or
9 mediators, one may develop more complex
10 models in terms of being able to model this
11 to come up with risk -- accurate risk
12 assessments.

13 But, again, this all has to be
14 within the context of the particular
15 condition, and this manuscript is written
16 very generically in the guidance.

17 **Q. Is there a GWAS model for**
18 **autism that accounts for environmental**
19 **confounding right now that we can look at?**

20 A. Again, what we have tried to
21 do, knowing that this is complicated, and
22 there's so many different subtypes, if you
23 will, of autism, so many different ancestral
24 backgrounds, we have tried to collect as much
25 detailed information across as many

1 individuals as possible to be able to power
2 to have such studies done.

3 I would say it's still a work
4 in progress, both in terms of recruiting
5 enough individuals, having enough exposure
6 data, having enough genotypic data.

7 But so far, based on currently
8 powered studies, we do not see the robust
9 effect of those environmental exposures,
10 especially once accounting for the genetic
11 factors.

12 Q. Well, but you don't have the
13 power to see anything yet?

14 A. So, as I said, I have not seen
15 within the context of being able to control
16 for the genetics seeing robust, reliable
17 exposure data.

18 Q. Yeah. I'm trying to understand
19 how you will ever accurately account for
20 environmental exposures in a population.

21 A. So I don't entirely know the
22 answer. That is part of what is up to the
23 collective scientific community --

24 Q. Okay.

25 A. -- to understand from a study

1 design point of view with different
2 populations around the world who have access
3 to different ways of measuring things
4 throughout the life course and looking at
5 outcomes with the constraints of the fact
6 that research takes money and whether or not
7 individuals think this is a priority to fund
8 and what diseases they decide or conditions
9 they decide to fund this for.

10 Q. Okay.

11 A. So it may be scientifically
12 possible. It will be a societal decision to
13 decide in terms of priorities.

14 Q. Okay. The next thing they put
15 on their list under the heading of "Foster
16 Robustness and Scientific Development,
17 Validation and Application and Interpretation
18 of PGS," second on this list is the comment
19 that "most PGS calculations assume a lack of
20 interaction and correlation between genetic
21 and nongenetic factors.

22 "Such genotype-by-environment
23 interactions and correlations create two
24 major limitations for the use of PGSS; a
25 difference in environmental backgrounds

1 **between the GWAS cohort and target**
2 **individuals can have unpredictable effects on**
3 **PGS accuracy and biases; and 2, the**
4 **interaction and correlation between a PGS and**
5 **environmental variables can induce a collider**
6 **bias that undermines the use of the PGS as a**
7 **covariant in modeling a variety."**

8 **You agree with that?**

9 A. Again, you have to get to the
10 specificity of what the trait is, and in this
11 manuscript, they have not listed the traits
12 that they believe are specifically at risk
13 for these types of concerns that they have.

14 **Q. Well, they say they all are.**
15 **They say "most," right?**

16 A. In fact, they say that as a
17 generic way, but they -- there are different
18 conditions that will have different relative
19 contributions of genes and environment.

20 And so there's certain
21 conditions that they're more concerned about
22 where that heritability and those risk
23 estimates of heritability are lower.

24 **Q. But as we sit here today, you**
25 **can't point me to any ASD PGS score that**

1 **accounts for the environment; it doesn't**
2 **exist?**

3 A. So right now, as we've spoken
4 about before, we're still in very early
5 stages of the accuracy of any of the
6 polygenic risk scores for autism. That's not
7 to say that genetics is not playing a key
8 role and that heritability is not extremely
9 high.

10 It's to say that our sample
11 size to be able to see those variants that
12 are statistically significant and
13 contributing is still limited.

14 **Q. And the same is true of ADHD?**

15 A. We are more advanced in our
16 understanding of the polygenic risk of ADHD
17 than we are for autism, but it suffers many
18 of the same limitations.

19 **Q. And so any study that seeks to**
20 **rely on polygenic risk scores to account**
21 **for -- to claim confounding by genetics is**
22 **unsound scientifically?**

23 MS. BROWN: Objection to the
24 form.

25 THE WITNESS: You may be

1 misunderstanding I think how people
2 are using the PRSs in these studies.

3 I think if they're able to
4 account for something, they truly have
5 accounted for it, because all of these
6 are always on the conservative side.

7 Rather, when you aren't able to
8 account for this, it doesn't rule out
9 the possibility that there might be
10 genetic confounds that are not
11 accounted for within those studies.

12 QUESTIONS BY MR. TRACEY:

13 **Q. No, Doctor.**

14 **If somebody claims that a**
15 **polygenic risk score for autism or ADHD is an**
16 **accurate reflection of the genetic risk**
17 **and ignores the environmental risk, that is**
18 **unsound scientifically, and we can't do that**
19 **yet; scientifically it's not possible to do**
20 **that yet?**

21 MS. BROWN: Objection to the
22 form.

23 THE WITNESS: So I want to
24 clarify because I think there are two
25 different things that we're talking

1 about.

2 Our estimates of polygenic risk
3 score currently are not accounting for
4 all the genetic factors that we see.
5 That results in some of the
6 imprecision, but it does account for
7 some of the genetic variables.

8 So they will account for some
9 of the variation that we see in terms
10 of the behavior of the phenotype, in
11 this case autism or ADHD.

12 When we see that contribution,
13 it is truly there, but there may be
14 other contributing factors as well.

15 QUESTIONS BY MR. TRACEY:

16 Q. Right.

17 And if you don't know the other
18 contributing factors, right --

19 A. You are underestimating the
20 genetic factors.

21 Q. I don't think so, Doctor,
22 because what you're ignoring is the part
23 where they say most PGS calculations assume a
24 lack of interaction between the genes and the
25 environment.

1 A. So --

2 **Q. That's what's going on here in**
3 **these -- in these studies that presume to**
4 **give an accurate polygenic risk score.**
5 **That's why they're -- that's why you said**
6 **they're of no clinical use.**

7 A. They're of no clinical use
8 today.

9 **Q. Yes.**

10 A. And that's a very specific use
11 case. That does not mean they're of no
12 scientific use. They can be of scientific
13 use to be able to understand in complex
14 models with large numbers of people what
15 percent of the variation within that
16 population, for instance, is due to the
17 genetic factors or if we can understand in
18 particular cases whether an outcome is in
19 part due to genetics or whether it's due to
20 an exposure.

21 What I'm stating is that you
22 can't not account for the genetics. Given
23 how high the heritability of the conditions
24 of autism and ADHD are, you will be
25 potentially confounded by other factors if

1 you don't include the genetics.

2 Especially if the exposure has
3 a relatively low risk associated with it, it
4 is even more susceptible to confounding, and
5 it is even more important to take into
6 account in whatever way possible those
7 confounding factors, some of which are
8 genetic, but others of which may not be
9 genetic.

10 **Q. May be what? Say it.**

11 A. I don't know.

12 **Q. Say "environment." You can say**
13 **it.**

14 A. I don't know.

15 **Q. Well, environment, that's one**
16 **of them, right?**

17 A. My point is --

18 **Q. Say environment first.**

19 MS. BROWN: Hold on. Wait.
20 Wait. Hold on. Let her answer,
21 please.

22 THE WITNESS: Again, for a
23 condition or conditions, ADHD and
24 autism, that are so strongly
25 heritable, one needs to account for

1 that in trying to account for the
2 outcomes and any other associations
3 with the outcomes.

4 QUESTIONS BY MR. TRACEY:

5 **Q. We're going round and round.**
6 **The problem is your definition of heritable**
7 **is not the definition any of my geneticists**
8 **use because your definition of heritable**
9 **assumes, as I understand it the way you keep**
10 **repeating it, no environmental contribution.**

11 And that's inconsistent with
12 what I understand to be the definition of
13 heritable.

14 So every time you say "with the
15 heritability being so high," I hear you
16 presuming no environmental contribution?

17 MS. BROWN: That totally
18 misstates her testimony for the past
19 seven hours.

20 MR. TRACEY: Am I wrong in
21 doing that?

22 MS. BROWN: Yes. Yes. Let her
23 explain it again carefully because I
24 think you'll get it.

25

1 QUESTIONS BY MR. TRACEY:

2 Q. Speak slowly. I'm not very
3 bright, Doctor.

4 MS. BROWN: Give -- give --

5 THE WITNESS: I'm sure you're
6 very bright.

7 MS. BROWN: Give him the
8 percentages so he understands.

9 QUESTIONS BY MR. TRACEY:

10 Q. Well, the percent -- I get the
11 percentages, but the percentages --

12 A. So --

13 Q. When you say heritable is 80 to
14 90 percent -- I mean, this -- we've been
15 reading this all day long.

16 That assumes -- this is the
17 overinflation of genetics. It's the
18 overinflation of the genetic contribution,
19 which is why I asked the question about when
20 you're a hammer, everything looks like a
21 nail, right?

22 A. So in multiple studies that
23 have looked at heritability for both
24 conditions, autism as well as ADHD, there is
25 a range in terms of that heritability, but it

1 is never 100 percent.

2 By that -- with those data, I
3 think I am stating and I have stated a few
4 times now today, that I don't believe it is
5 100 percent genetic.

6 As we think about the
7 contributions in a population of individuals,
8 a number of individuals, knowing that autism
9 is a spectrum, is quite heterogenous, for
10 autism or for ADHD, it is still
11 overwhelmingly genetic.

12 But --

13 **Q. What does that mean?**

14 A. That means that for -- if one
15 is to look at the variation in the phenotype
16 in a large population, the predominance of
17 the variation is attributable to genetic
18 factors.

19 Be they inherited or de novo
20 factors, it is still genetics that is
21 overwhelmingly driving the phenotype.

22 **Q. But this is where I think you**
23 **and I are driving past each other.**

24 You proved in a paper that
25 de novo mutations can be caused by

1 **environmental exposures, right?**

2 A. In the paper?

3 **Q. Yes, ma'am.**

4 A. It is not looking at human
5 people and what exposures --

6 **Q. What kind of people is it**
7 **looking at?**

8 A. -- in human people, living
9 human people. It is not showing in living
10 human people --

11 **Q. It's a cell study?**

12 A. -- what is -- what is driving
13 de novo mutations in living human people that
14 may be associated with autism.

15 **Q. Okay. But my point is, the way**
16 **I think that we're passing each other is --**
17 **and maybe I'm mishearing you or**
18 **misunderstanding you, which I'm completely**
19 **capable of.**

20 You seem to be assuming that
21 the environment has no direct effect or even
22 indirect effect on the genetics of human
23 beings living in an environment?

24 A. I don't believe I've stated
25 that.

1 Q. Okay.

2 A. Right.

3 Q. Good.

4 So, but if you don't know what
5 environmental exposures are that are causing
6 the different ways that genetics can be
7 impacted, and you don't account for that, and
8 you presume that everything is genetic and
9 genetic only, you're reaching a foreordained
10 conclusion.

11 You're a priori deciding
12 without evidence that everything is genetic
13 when we know the environment influences a
14 common variance. We know that. We know that
15 it influences de novo mutations.

16 A. What I'm stipulating --

17 MS. BROWN: I object to the
18 question on a number of grounds,
19 including lacking foundation and
20 compound.

21 Go ahead.

22 THE WITNESS: What I'm
23 saying -- and I realize that it's hard
24 to prove these things. I admit, these
25 are hard questions. Biology is hard.

1 Human behavior is hard to understand.

2 And to be able to implicate any
3 of those environmental exposures, one
4 must account for the genetics in the
5 equations of those environmental
6 exposures because genetics is so
7 strong overall in a population of
8 individuals.

9 And what I have not seen in the
10 studies that implicate any of those
11 environmental exposures for autism or
12 ADHD is an appropriate study design
13 that can include genetics as part of
14 the equation.

15 So even if you are correct in
16 terms of those gene-by-environmental
17 exposures in your model, you would
18 still, I think, argue that one would
19 need to include the genetics in those
20 study designs to be able to account
21 for those interactions.

22 QUESTIONS BY MR. TRACEY:

23 Q. Yes. Okay.

24 Science is hard, isn't it?

25 A. Science is hard, and people are

1 hard.

2 (Chung Exhibit 305A marked for
3 identification.)

4 QUESTIONS BY MR. TRACEY:

5 Q. Oh, yes. This will be my last
6 thing because I want to get out of here
7 quickly. This is a real last thing, not a
8 lawyer last thing.

9 On the break -- at the
10 beginning of the deposition -- I'm going to
11 sort of come back to where I began.

12 You -- I asked you about
13 methodology, any methodology that you could
14 articulate in your paper, and we had that
15 discussion.

16 Do you remember that?

17 A. Yes, I do.

18 Q. And then I asked you whether in
19 your professional life, outside of
20 litigation, you actually employed
21 methodologies when you were doing analyses of
22 data and scientific information.

23 Do you remember that?

24 A. Yes, I do.

25 Q. And so on the break, somebody

1 found this paper where it seems that you've
2 done just that. This was a paper published
3 in 2019 in Genetics in Medicine. It's called
4 "A Systematic Review." And you are one of
5 the authors.

6 Are you not?

7 A. Yes, I was.

8 Q. And then just flip over to the
9 next page, you have "Materials and Methods."

10 Do you see that?

11 A. Yes, I see.

12 Q. You said you conducted -- or
13 we, not you. "We conducted a scoping review
14 as the basis for a consensus development
15 conference to summarize the molecular
16 diagnostic yield of ES for NDDs, specifically
17 compared with CMA. For the conference, in
18 addition to the core group of experts who
19 authored this paper, we also invited outside
20 experts on the CNV analysis from ES data."

21 The details don't matter to me
22 much, but the next paragraph does.

23 In the scope -- you said, "For
24 this scoping review, we addressed the
25 following question."

1 And then you -- you tell us the
2 question you're addressing, don't you?

3 You say, "Among individuals
4 with NDDs tested with ES, what is the
5 molecular diagnostic yield compared with
6 CMA?"

7 So you've identified and
8 articulated the question that you were
9 addressing in your paper, haven't you?

10 A. Yes.

11 Q. Then you say, "We selected
12 articles from PubMed focusing on ES and NDDs.
13 We included studies that involve sequencing
14 of protein-coding, regions of Mendelian genes
15 and excluded studies that were gene panels
16 sub-sampled from exome data. We defined NDD
17 as GDD, ID and/or ASD. We searched PubMed
18 with a combination of medical subject
19 headings, terms and keywords pertaining to
20 NDDs."

21 And then you give examples, and
22 then you give the date of your search,
23 January 2014 to June 2018.

24 Then you say, "A scoping review
25 differs from the systematic reviews in that

1 **their focus is on more broadly defined**
2 **research questions, charting of themes and**
3 **development of inclusion/exclusion criteria**
4 **at the study selection stage."**

5 **Did I read all of that**
6 **correctly?**

7 A. Yes, you've read that
8 correctly.

9 Q. And in this systematic review,
10 you identified for everybody the question
11 that was being asked, and then you
12 articulated the search terms and how you went
13 about identifying the literature that you did
14 identify that you were setting out to review.

15 Correct?

16 A. Yes, that's correct.

17 Q. None of that was done in your
18 report?

19 MS. BROWN: Objection to the
20 form.

21 THE WITNESS: This review that
22 was done was done for a very specific
23 reason and indication, which is that
24 as we're trying to assess clinical
25 guidelines in this case, diagnostic

1 yields for neurodevelopmental
2 disorders. It was very clear how to
3 be able to look at the published data
4 for this and largely to be able to
5 help guide clinicians and payers in
6 what tests should be performed.

7 This is quite a different
8 process than what I was asked to do in
9 terms of developing the opinions for
10 today.

11 This was not dissimilar in
12 terms of doing a literature review
13 with key search terms and being able
14 to look at the evidence that was in
15 those manuscripts and summarizing that
16 data in one comprehensive source.

17 I did, when I was using the
18 methods after performing the
19 literature review, consider many
20 things, many things in terms of the
21 sample size of the papers that I
22 reviewed, the replicability across
23 studies, the effect size that was
24 seen, the rigor of testing and
25 penalizing for multiple statistical

1 tests. So I did apply that method,
2 although it was different from this
3 method because this was a different,
4 focused clinical question than I was
5 asked to review for my opinion.

6 QUESTIONS BY MR. TRACEY:

7 **Q. The answer to my question is,**
8 **what we have on the page there that you did,**
9 **identifying a question and then all your**
10 **search terms, is absent from your report.**

11 MS. BROWN: Objection to the
12 form. Misstates her testimony.

13 QUESTIONS BY MR. TRACEY:

14 **Q. Right?**

15 A. Again, we went through it
16 earlier this morning. Within my report, I
17 did demonstrate the area which was the key
18 questions, the scope of what I was
19 addressing.

20 **Q. Can you show me in your report,**
21 **ma'am, where -- like you have on the page**
22 **here in this published paper, the question**
23 **that you were answering like we see it in**
24 **this paper?**

25 A. Again, we referenced this this

1 morning, but on pages 2, 3 and 4, we have the
2 questions and the opinions. They're not
3 framed in the form of a question, but we do
4 have my opinions listed there.

5 I did not include the search
6 terms in my report, but I stated this
7 morning, as we were discussing it, how I
8 performed the PubMed literature search.

9 Q. I just -- one more question.

10 In your disclosures to us, you
11 did not identify a case where you gave a
12 deposition in July of 2022 in a case called
13 Hermiller {phonetic} versus Mary -- Mercy,
14 sorry.

15 Do you know why you didn't
16 disclose that as a deposition you've given in
17 the last four years?

18 A. It was an oversight if I didn't
19 put -- include it.

20 Q. Okay. The other -- okay.
21 Okay. So it should -- it says four cases
22 you've testified in as an expert witness in
23 the last four years, but it should be five.

24 You remember the Mercy case,
25 right?

1 A. I don't off the top of my head.

2 **Q. I read the deposition. I think**
3 **it was a med-mal case.**

4 A. Okay.

5 **Q. In Philly.**

6 A. Okay.

7 **Q. Do you remember that now?**

8 A. I'm sure it was a med-mal case,
9 yes.

10 **Q. How many depositions have you**
11 **given in your lifetime?**

12 A. In my lifetime, if that was
13 five and today's six, then seven and eight
14 and nine.

15 **Q. Nine.**

16 **Okay. Have you ever testified**
17 **at trial?**

18 A. Once.

19 **Q. And what kind of -- is that --**
20 **you were an expert in that case?**

21 A. Yes.

22 **Q. What kind of case was that?**

23 A. Medical malpractice.

24 **Q. Where was that?**

25 A. It was a very long time ago, in

1 New York.

2 MR. TRACEY: Okay. Okay.

3 What's that? Oh, they want me to mark
4 your bills as an exhibit and attach
5 them. And let's mark the depo notice,
6 do the cleanup stuff.

7 Doctor, thank you for your
8 time. I don't have any further
9 questions. I appreciate it.

10 Have I been reasonably nice to
11 you?

12 THE WITNESS: It's been an
13 interesting day.

14 MR. TRACEY: Yeah. For me,
15 too, thanks.

16 I'll pass the witness.

17 (Chung Exhibits 301 and 303
18 marked for identification.)

19 MS. BROWN: I have just a
20 couple of questions.

21 CROSS-EXAMINATION

22 QUESTIONS BY MS. BROWN:

23 Q. Dr. Chung, are you ready just
24 to answer --

25 A. Sure.

1 Q. -- a few questions.

2 Okay. Counsel just showed you
3 an article titled "Meta-analysis and
4 multi-disciplinary consensus statement."

5 MS. BROWN: Did we mark this as
6 an exhibit?

7 MR. TRACEY: Oh, no. We
8 should.

9 MS. BROWN: Okay. Do we have a
10 number?

11 Ah. It's been marked as 305A.

12 MR. TRACEY: Oh, okay. So I
13 don't think I said that.

14 QUESTIONS BY MS. BROWN:

15 Q. Okay. And, Dr. Chung, I want
16 to direct your attention to the second page
17 of Exhibit 305A. Look with me, if you would,
18 at the left-hand column, the very last
19 paragraph that begins "Therefore."

20 Do you see that?

21 A. Yes.

22 Q. And what your paper that we
23 marked as Exhibit 305A states is that
24 "Therefore, our primary objective was to
25 conduct an evidence-based consensus

1 conference to provide recommendations for the
2 use of ES in the diagnostic evaluation of
3 individuals with NDDs."

4 Do you see that, Doctor?

5 A. Yes, I do.

6 Q. Okay. Was the primary
7 objective of your expert report that we've
8 marked as 305 the same?

9 A. No, it was different. This
10 was, as I said, designed to be able to inform
11 diagnostic evaluations.

12 Q. And counsel pointed you to a
13 section of 305A that discussed something
14 called a "scoping review."

15 Do you remember looking at that
16 language?

17 A. Yes.

18 Q. And is that something that you
19 did as part of your report?

20 A. I didn't call it a scoping
21 review, per se, but I considered them both
22 literature reviews that are comprehensive in
23 scope of defining the question and reviewing
24 all the relevant literature.

25 Q. And counsel asked you, well,

1 where can we find the question in your
2 report? And you directed him to, in part,
3 pages 2, 3, 4 of your report, correct?

4 A. Correct.

5 Q. And I want to draw your
6 attention to paragraph number 2, Dr. Chung,
7 where you state, "This report identifies the
8 robust and rigorously replicated scientific
9 evidence in the published literature
10 demonstrating the known genetic etiologies of
11 ASD and ADHD."

12 Do you see that, Doctor?

13 A. Yes, I do.

14 Q. And what was the intent of that
15 sentence in your report at page 2,
16 paragraph 2?

17 A. The intent was to identify what
18 the scope of this report was to include.

19 Q. And is that the same
20 methodology that you apply when you were
21 publishing one of your 700 papers?

22 A. The same in the sense of
23 being -- doing a comprehensive literature
24 review and summarizing all of that literature
25 and -- anyway, being able to represent it

1 accurately and concisely.

2 Q. And the next sentence in
3 paragraph 2 says, "My report also addresses
4 the published epidemiological studies on
5 prenatal acetaminophen exposure and outcomes
6 purportedly relating to ASD and/or ADHD to
7 assess whether these studies have properly
8 accounted for genetic confounders."

9 Do you see that sentence?

10 A. Yes.

11 Q. And what was the intent of that
12 sentence in paragraph 2, page 2 of your
13 report?

14 A. As we've been discussing today,
15 it was to be able to understand specifically
16 the context of acetaminophen and prenatal
17 exposures and to understand whether genetic
18 factors have been accounted for in those
19 epidemiological studies.

20 Q. And did you apply the same
21 methodology that you apply in your published
22 papers to your efforts to do what you've --
23 what you've set forth in paragraph 2 of your
24 expert report?

25 A. Yes, I tried to use the same

1 scientific methodology and literature review
2 that I would do in any of my other scientific
3 work.

4 Q. So if someone were to take
5 Exhibit 305A and line it up with your expert
6 report, which we've marked as 305, and
7 suggest that you somehow have employed a
8 different methodology outside of litigation
9 than you've employed as an expert witness in
10 litigation, would that be fair?

11 A. I would say I haven't
12 specifically spelled it out in the same way
13 that we do in a methods paper by calling it a
14 scoping review, per se, but it's the same
15 process and the same literally PubMed search
16 engine that's used for both of the literature
17 reviews and assessments.

18 Q. So have you done anything
19 different outside of litigation than what
20 you've done inside of litigation when it
21 comes to creating your expert report that
22 we've marked as 305?

23 A. No, I'm scientifically trained
24 to perform scientific analyses the most
25 rigorous way I know how.

1 Q. And did you apply the same
2 scientific rigor to your expert report that
3 we've marked as Exhibit 305 that you've
4 applied to your many publications that you
5 have throughout the years?

6 A. I believe so. I believe I've
7 been very careful and very rigorous in my
8 report.

9 MS. BROWN: Thanks, Dr. Chung.
10 I have no further questions.

11 THE WITNESS: Thank you.

12 MR. TRACEY: I don't have
13 anything else, Dr. Chung. Thank you.

14 THE WITNESS: Thanks.

15 MS. BROWN: Okay.

16 VIDEOGRAPHER: With that, the
17 time is 4:16 p.m. This deposition has
18 concluded, and we're off the record.

19 (Deposition concluded at 4:16 p.m.)

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Confidential Subject to Protective Order

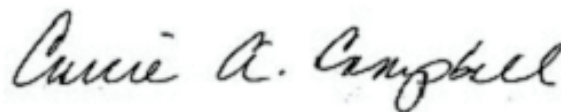
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CERTIFICATE

I, CARRIE A. CAMPBELL, Registered Diplomate Reporter, Certified Realtime Reporter and Certified Shorthand Reporter, do hereby certify that prior to the commencement of the examination, Wendy Chung, MD, Ph.D., was duly sworn by me to testify to the truth, the whole truth and nothing but the truth.

I DO FURTHER CERTIFY that the foregoing is a verbatim transcript of the testimony as taken stenographically by and before me at the time, place and on the date hereinbefore set forth, to the best of my ability.

I DO FURTHER CERTIFY that I am neither a relative nor employee nor attorney nor counsel of any of the parties to this action, and that I am neither a relative nor employee of such attorney or counsel, and that I am not financially interested in the action.



CARRIE A. CAMPBELL,
NCRA Registered Diplomate Reporter
Certified Realtime Reporter
California Certified Shorthand
Reporter #13921
Missouri Certified Court Reporter #859
Illinois Certified Shorthand Reporter
#084-004229
Texas Certified Shorthand Reporter #9328
Kansas Certified Court Reporter #1715
New Jersey Certified Court Reporter
#30XI00242600
Louisiana Certified Court Reporter
#2021012
Notary Public
Dated: August 31, 2023

1 INSTRUCTIONS TO WITNESS

2
3 Please read your deposition over
4 carefully and make any necessary corrections.
5 You should state the reason in the
6 appropriate space on the errata sheet for any
7 corrections that are made.

8 After doing so, please sign the
9 errata sheet and date it. You are signing
10 same subject to the changes you have noted on
11 the errata sheet, which will be attached to
12 your deposition.

13 It is imperative that you return
14 the original errata sheet to the deposing
15 attorney within thirty (30) days of receipt
16 of the deposition transcript by you. If you
17 fail to do so, the deposition transcript may
18 be deemed to be accurate and may be used in
19 court.

ACKNOWLEDGMENT OF DEPONENT

I, _____, do
hereby certify that I have read the foregoing
pages and that the same is a correct
transcription of the answers given by me to
the questions therein propounded, except for
the corrections or changes in form or
substance, if any, noted in the attached
Errata Sheet.

Wendy Chung, MD, Ph.D. DATE

Subscribed and sworn to before me this
_____ day of _____, 20 _____.
My commission expires: _____

Notary Public

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